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A NEW SURGICAL TECHNIC FOR THE TREATMENT OF POSTPHLEBITIC VARICOSE ULCERS OF THE LOWER LEG

A Preliminary Report

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THE distressing sequela of ulceration of the lower leg following deep phlebitis has been a problem in medicine for many years. Numerous types of treatment have been recommended. The majority of these have been temporary healing measures. In addition several surgical procedures have been advocated, notably by Homans[4, 5] and Trout.[9] The most commonly employed of these methods has been that of Homans. It consists in the excision of the ulcerated area with a wide margin of the thick, indurated and edematous skin and subcutaneous tissues about it. The dissection is carried through the deep fascia down to normal muscle. An immediate skin (Thiersch) graft is placed over the denuded area.

Many ulcers have been healed by this procedure, but the results from a cosmetic point of view often are not all that is to be desired. In addition, a number of them have not been healed by this method. Because of this it was thought advisable to develop some new procedure that would improve the end results.

A careful study of the underlying lesion seemed essential to a satisfactory solution. It was noted that a postphlebitic varicose ulcer could be readily cleaned up by putting the patient to bed and applying moist compresses of boric acid solution, normal saline or a surgical solution of chlorinated soda. After the granulation tissue in the ulcer bed had taken on a normal red color it was always possible to do a successful skin (Thiersch) graft. In fact the grafts always made extraordinarily good takes, and flourished as long as the patient was kept in bed. Soon after the patient became ambulatory, however, it was very often found that the skin grafts rapidly disintegrated. The question naturally arose, Why did the graft remain intact as long as the patient was in bed, but melt away after he was allowed up and about? The usual answer was that there was so much scar tissue beneath the ulcer that the new skin did not get sufficient nourishment to survive. Since the skin graft always remained perfectly intact if the patient was kept in bed, this did not seem to be the correct answer. Further analysis of the condition suggested that the underlying cause lay in the veins which connect the deep venous systems of the leg with the superficial ones. These are known as the communicating veins but are sometimes called the perforating veins.

In the normal leg these veins possess valves which allow the blood to flow only from the superficial to the deep systems. Following deep phlebitis, it has been recognized for a long time that the valves become incompetent, allowing blood to pass in either direction. Since this is an abnormal condition, exposing the superficial system of veins directly to the pressure of the deep veins, it was thought that this might be a factor in the persistence of postphlebitic varicose ulcers. In support of this theory recent studies by Holling, Beecher and Linton[8] have shown that in the presence of incompetent communicating veins there is considerably more edema formation in the lower leg than when only the saphenous veins are incompetent.

A careful search of the surgical and anatomical literature failed to reveal any detailed studies of these communicating veins. In fact the most accurate description was that of a Russian anatomist, von Loder,[7] who published his work in 1803. Because of this dearth of knowledge careful anatomical dissections were made in ten amputated legs. The superficial, deep and communicating venous

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systems were injected with a suspension of barium sulfate in water. The water gradually left the vessels, the white barium sulfate remaining in them. The veins stood out in white so that they were readily recognizable in the tissues of the leg. The

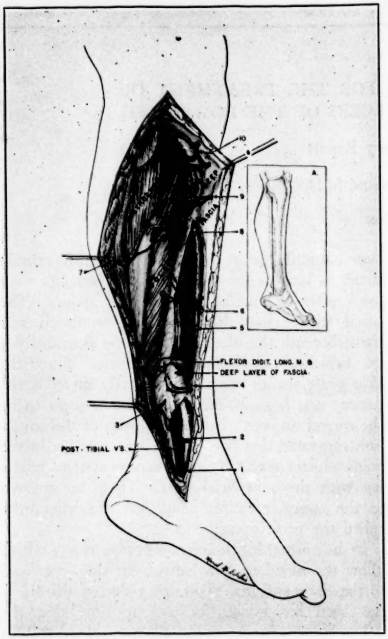

Figure 1. (Reproduced by courtesy of the *Annals of Surgery* [*107*:582, 1938].) *The medial group of communicating veins of the lower leg. A dissection of the medial aspect of the lower leg to show the medial group of communicating veins. The dissection has been carried out beneath the deep fascia. In the lower part of the leg the lamina profunda of the deep fascia has been divided to show the origin of the lower communicating veins. Numbers 1, 2, 3, 4, 6, 8 and 9 are posterior tibial communicating veins. Numbers 5 and 8 belong to the medial subdivision of the anterior tibial communicating veins. Number 7 represents a perforating vein which drains blood from the muscles through the deep fascia to the superficial veins. The inset shows the location of the incision used in the operation for the ligation of these veins.*

results of this study have been published in a recent article.[6] The reader should refer to this for a more detailed description. In brief, it was found that for practical purposes the communicating veins could be divided into three main surgical groups: the medial, the anterior and the lateral. The medial group is found on the inner aspect of the lower leg and is made up chiefly of the posterior tibial communicating veins which connect the posterior tibial veins with the long and short saphenous veins and their tributaries; in addition there is the medial subdivision of the anterior tibial communicating veins (Fig. 1). The anterior group is found on the anterolateral aspect of the lower leg. It consists of the central and lateral subdivisions of the anterior tibial communicating veins which connect the anterior tibial veins with the superficial ones (Fig. 2). The lateral group is made

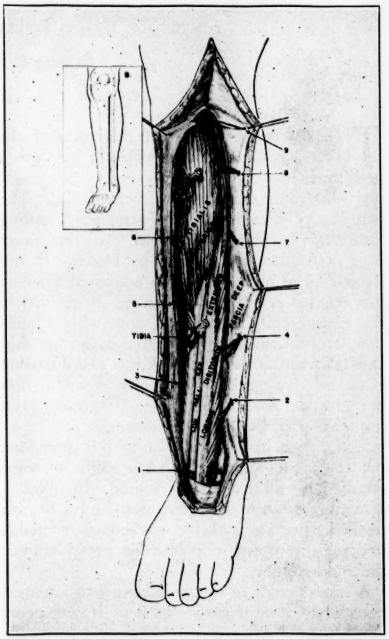

Figure 2. (Reproduced by courtesy of the *Annals of Surgery* [*107*:582, 1938].) *The anterior group of communicating veins of the lower leg. A dissection of the anterior aspect of the lower leg to show the anterior group of communicating veins beneath the deep fascia. Numbers 1, 2, 4, 7, 8 and 9 are the lateral subdivisions of this group emerging along the anterior peroneal intermuscular septum. The tibialis anticus muscle is retracted laterally to show the veins of the central subdivision, Numbers 3, 5 and 6 on the lateral aspect of the tibia. The inset shows the location of the incision used in the operation for the ligation of these veins.*

up of the peroneal communicating veins. These connect the peroneal veins with superficial ones, chiefly those draining into the short saphenous vein (Fig. 3). In addition to these three groups there

is another fairly constant communicating vein which arises from the popliteal vein, descending between the heads of the gastrocnemius muscle. About the middle of the lower leg it pierces the deep fascia to join the short saphenous vein (Fig. 3).

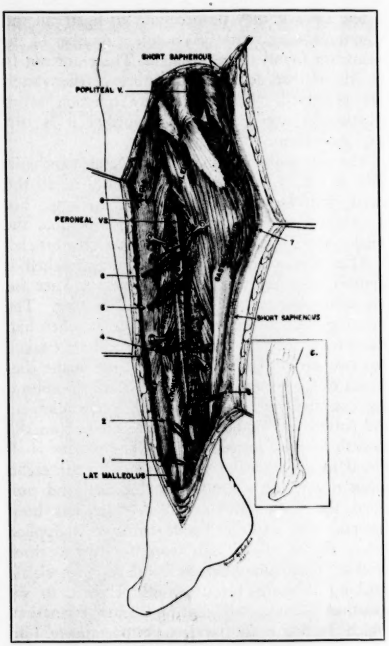

Figure 3. (Reproduced by courtesy of the *Annals of Surgery* [*107*:582, 1938].) *The lateral group of communicating veins of the lower leg. A dissection of the posterolateral aspect of the lower leg to show the lateral communicating veins as they arise from the peroneal veins and pass outward along the posterior peroneal intermuscular septum. These are Numbers 1, 2, 3, 4, 5, 6 and 8. Posteriorly, the deep fascia has been elevated to show the popliteal communicating vein, Number 7. The short saphenous vein is shown superficial to the deep fascia. The inset shows the incision used in the operation for the ligation of these veins.*

The anatomical study revealed that all the veins could be exposed surgically through three straight longitudinal incisions (Figs. 1, 2 and 3). The incisions are carried through the deep fascia down to the muscles. The dissection between the muscles and the fascia is readily made since the only structures between them are the communicating veins, accompanied by small arteries and a loose areolar connective tissue. All these vessels emerge from the intermuscular spaces and pass through the deep fascia to join veins of the saphenous systems in the subcutaneous tissues. They are readily divided and ligated beneath the fascia.

Ligation of the perforating veins has been advocated for many years; Rémy[8] in 1901, Homans[4, 5] in 1916 and 1917 and Trout[9] in 1929 have stressed the importance of this fact. It is to be noted that an operation known as the "flap" operation for incompetent communicating veins was performed for a number of years at the Massachusetts General Hospital. It was not used primarily for postphlebitic varicose ulcers but in cases of varicose veins in which incompetent communicating veins were demonstrable. This operation consisted in the use of a short curved incision in the lower leg where the incompetent communicating veins were demonstrable. A flap of skin and fascia was formed, and was elevated in order to ligate the communicating veins. The operation fell into disrepute because not infrequently the skin edge of the flap sloughed, requiring weeks or months to heal. The longer straight incisions are an important improvement in this operation. Even though they are carried through healed ulcer areas, it has been possible to avoid sloughing of the wound edges. In addition, a much more complete exposure of the communicating veins is obtained.

The following plan of treatment is recommended in the handling of a postphlebitic varicose ulcer. When first seen the granulation tissue of the ulcer is frequently covered with a dirty-grayish membrane. Very often there is a thin, copious, purulent discharge. Cultures are taken before any dressings are applied. These usually show a mixed growth of bacteria; sometimes hemolytic streptococci are present, but most commonly staphylococci. The patient is placed in bed. A blood test is done to rule out syphilis. The leg with the ulcer is elevated on a pillow to the level of the heart or a little above it. Warm 2 per cent boric acid solution or saline compresses are applied to the ulcer area every two hours. If hemolytic streptococci are present, compresses of a surgical solution of chlorinated soda (Dakin's solution) are most efficacious. Under this regime the condition of the leg and of the ulcer rapidly improves. Many of the smaller ulcers epithelialize rapidly with this treatment. The larger ones often require skin (Thiersch) grafts in order to hasten their healing. Unless streptococci have been discovered, the ulcer is usually in a satisfactory condition for skin grafting at the end of a week. The skin graft requires ten days to two weeks for a satisfactory take. At the end of this time the patient prepares for getting out of bed by doing postural leg exer-

cises for three or four days. These exercises are a modification of those described by Buerger[2] and Allen[1] in that the period of elevation is longer than the period of dependency.

When the patient is up he must wear a supporting elastic bandage. One or two days before he is discharged home the long saphenous vein of the affected limb is ligated in the groin. Although this vessel may not appear to be varicose it is rare not to find it incompetent in the postphlebitic leg. Sometimes one of the communicating veins between the long saphenous vein and the deep veins in the thigh is incompetent. If such is the case this vessel should be ligated at the same time. It has been found best to have the ulcer healed before doing so, in order to avoid wound infections. This is because the incision is made in the groin where there are many lymph vessels and nodes which drain lymph from the leg, so that one runs the risk of wound infection if the ulcer is still open. If the short saphenous vein is involved it is ligated in the popliteal space.

When the patient is discharged home, usually at the end of two and a half to three weeks, an elastic adhesive bandage is applied directly to the skin from the toes to just below the knee. This is changed every two or three weeks. At the end of six weeks the patient returns to the hospital for the ligation of the communicating veins. It has been found important to allow this period to elapse after the ulcer has healed before the communicating veins are operated on. This is because of the danger of infection from the skin, which has been bathed in pus for many months. It is felt that after the ulcer has been healed for six weeks the skin is sufficiently clean to carry out the operation safely. In all the cases which have been done in this manner the wounds have healed per primam. The skin of the lower leg should be carefully cleansed preoperatively with soap and water. If there is scaling or itching, a fungicide is applied twice a day until it is in a satisfactory condition for operating.

Because of edema, infection and scar tissue, it is as a rule very difficult in the postphlebitic leg with ulcerations to demonstrate which group of communicating veins is incompetent. However, it can be said almost with certainty that given a patient with a previous history of deep thrombophlebitis who has an ulcer on the lower leg, there are incompetent communicating veins in close relation to it. Because of the difficulty in palpating these vessels, it is the location of the ulceration that guides one in deciding which group or groups of communicating veins should be ligated. If the ulcer lies on the inner aspect of the lower leg the medial group of communicating veins and the popliteal communicating vein are ligated. If it lies on the anterior surface of the leg it is usually necessary to ligate the anterior group also. When the ulcer is on the posterior or posterolateral surface the lateral group and the popliteal communicating veins are ligated, and in addition the short saphenous vein is ligated in the popliteal space. In a few cases it may be necessary to ligate all the communicating veins. As a rule a number of the veins are found to be normal. These are not ligated, so that actually a number of the vessels are preserved. If there is any question as to whether or not they are incompetent it is best to ligate them.

The operation is done under spinal anesthesia. Silk has been used as suture material in all the cases, with very satisfactory wound healing. For a detailed account of the operative technic the reader should refer to the author's recent article.[6]

After the operation a posterior plaster shell is applied from the toes to just below the knee for immobilization so as to aid wound healing. The dressing is done first, on the tenth day, when half the stitches are removed. The remainder are taken out two to four days later, depending on the condition of the wound. If it is well healed, postural leg exercises are again instituted before allowing the patient out of bed. These are begun on the twelfth to the fourteenth day. The patient is allowed to go home on the sixteenth to the eighteenth day if the wound is well healed and provided that only one group of veins has been ligated. An elastic adhesive bandage is applied before discharge. This is worn for two to three weeks. Thereafter a bender bandage or an elastic stocking is worn for a period of three to six months. This is to control edema formation, which is prone to develop postoperatively. In some cases it is necessary to obliterate the superficial veins with injections of a sclerosing solution; in others this may not be required, as the varicose veins shrink to normal size after their communications with the deep veins have been severed. After several months some patients are able to discard supporting bandages, while others continue to wear them, either because of persistent edema or because they have worn them for so many years that they are afraid to do without them.

The following case report is typical of the disease and the result obtained by the procedure which has been outlined.

CASE REPORT

G. M. S., a 43-year-old, married, American housewife, entered the Massachusetts General Hospital on April 15, 1937, complaining of varicose veins and an indolent ulcer of the right lower leg of 7 years' duration. After a cesarean section 18 years previously the patient had a deep thrombophlebitis of the right leg. This gradually subsided in about 4 months. Since that time the superficial

veins of the right leg had been engorged and she had been troubled with swelling of the leg. Seven years previously an ulcer developed on the inner side of the right lower leg following an injection for the varicose veins. During the next 5 years the patient received approximately 125 intravenous injections of a sclerosing solution into the veins of this leg in another clinic. The ulcer became smaller under these treatments, but it never completely disappeared. Following a cholecystectomy 2 years prior to her admission, when she was confined to bed 16 weeks, the ulcer healed. It recurred, however, shortly after getting up. One year previous to admission it was healed by the use of an Unna's paste boot. After removal of this, the ulcer soon reappeared. The remainder of the history is noncontributory.

Physical examination showed a well-developed, somewhat obese, middle-aged woman. General examination was essentially negative except for the findings in her legs. Both legs presented varicose veins of the long saphenous systems. On the right there were two shallow ulcerating areas about 4 cm. in diameter (Fig. 4). Surrounding the ulcers there was a zone of brownish-red pigmentation 4 or 5 cm. in width. There was a thin, slightly purulent discharge from the ulcers. A culture yielded *Staphylococcus aureus*. The blood Hinton test was negative.

Figure 4. *A photograph of the right lower leg of a forty-three-year-old woman showing a postphlebitic varicose ulcer of seven years' duration. Note the two ulcerations and the discolored pigmented skin surrounding them.*

The patient was put to bed. The right leg was elevated on two pillows. Warm saline compresses were applied to the leg every 2 hours. The ulcer cleaned up very rapidly under this régime. On April 20, or 5 days after admission, the long saphenous vein was ligated in the groin. It was not injected. The patient insisted on going home on the 5th postoperative day. As the ulcer was not healed she was instructed to remain in bed and to apply warm boric-acid solution compresses.

The patient was seen in the Peripheral Vascular Clinic of the Out Patient Department of the hospital on May 8, 12 days after her discharge. The ulcer was clean but not healed so that an elastic adhesive (elastoplast) bandage was applied directly over the ulcer from the toes to just below the knee. In addition 1.5 cc. of a sclerosing solution of quinine and urethane was injected into the saphenous vein in the thigh.

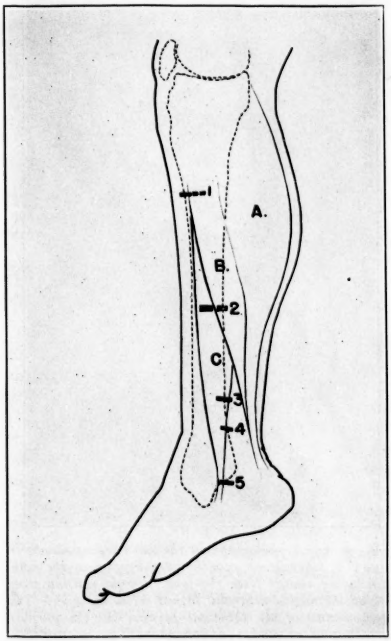

Figure 5. *A diagram showing the location of the posterior tibial communicating veins, Numbers 1, 2, 3, 4 and 5, found at operation. It is to be noted that the popliteal communicating vein was not ligated in this case. Since operation it has had to be injected twice. This vein in subsequent cases is always ligated if present. The gastrocnemius, soleus and flexor digitorum longus muscles are represented respectively by the letters A, B and C.*

Three weeks later, on May 28, the elastic adhesive bandage was removed and a new one was applied. The ulcer was healed at this time, but it was noted that the skin over and around the ulcer area was distinctly bluish. Another injection, 2 cc. of a solution of quinine urethane, was made into the saphenous vein in the thigh.

The patient was readmitted to the hospital for ligation of the medial group of communicating veins on June 23, approximately 9 weeks after the ligation of the long saphenous vein and 4 weeks after the ulcer had healed.

On June 26 the medial group of communicating veins of the right lower leg were ligated under spinal anesthesia.

Five sets of communicating veins were found and ligated, as shown in Figure 5. The largest ones were in the lower third of the leg. The popliteal communicating vein was not ligated in this case, as the importance of this vein was not recognized at operation. The postoperative convalescence was uneventful and the wound healed per primam. The patient was discharged home 14 days postoperatively. An elastic adhesive bandage was applied directly to the skin from the toes to just below the knee to control postoperative edema that is apt to develop. This bandage was removed in 3 weeks, after which she wore an elastic bender bandage during the day for the next 3 months.

Figure 6. *A photograph of the same leg as shown in Figure 4, taken seven months after ligation of the communicating veins. Note the long, straight incision from behind the medial malleolus to just below the knee. The pigmentation of the skin still persists, but the purplish discoloration of it has disappeared. The whitish area in the center is the location of the healed ulcerations. There is slight tendency toward keloid formation in the scar. This is not characteristic of all cases. The scar as in this one is narrowest in the region of the ulcer area. This is characteristic of all the cases.*

The photographs (Figs. 6 and 7) were made February 5, 1938, or 7 months after the ligation of the communicating veins. They show the site of the well-healed ulcer. The patient had worn no supporting bandage for the previous 3 months. She said that she noticed a little edema of the right lower leg at the end of the day at times, but that the ulcer remained healed. Examination showed a well-healed ulcer. There was still a residual pigment discoloration in the skin, which had diminished slightly. The saphenous vein was palpable and patent below the knee, as was also the popliteal communicating vein in the mid-posterior calf region. An injection of 1.5 cc. of quinine urethane solution was made in the saphenous vein. The patient was last seen March 25, 1938, 9 months postoperatively, at which time the leg was still healed and in excellent condition.

Comment. This is a typical case of postphlebitic varicose ulcer appearing eleven years after a deep thrombophlebitis of the leg following a cesarean section. Despite numerous injections of sclerosing solutions into the veins, the ulcer persisted. It characteristically healed completely during a sixteen-week confinement to bed following a cholecystectomy, but recurred soon after getting up. It was healed again by the use of an Unna's paste boot, but again recurred when the boot was removed. Following ligation of the long saphenous vein and the medial group of communicating veins of the lower leg and obliteration of the long saphenous vein with injections of quinine urethane solution, the ulcer has remained healed for nine months. During the last five months of this period the pa-

Figure 7. *A close-up photograph of the healed ulcer to show the condition of the skin and operative scar in the old ulcer area. This was taken February 5, 1938, seven months after ligation of the communicating veins. Four months later, at the time of this writing, the ulcer was still healed.*

tient has not worn a supporting bandage or elastic stocking. The result is not perfect, as there is a slight amount of edema at the end of the day, but it can certainly be classed as satisfactory.

A series of 37 postphlebitic varicose ulcers of the lower leg were operated on during 1937 according to the above technic. The results are to be published in another article as soon as sufficient time has elapsed to permit an adequate follow-up.

SUMMARY

A new type of surgical treatment of postphlebitic varicose ulcers is described.

The importance of the communicating veins of the lower leg in the etiology of these ulcers is stressed.

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OSTEOPOROSIS IN HYPERTHYROIDISM

Reports of Two Cases with Compression Fractures of the Vertebrae

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BOSTON

CHANGES in the bones have been known to result from hyperthyroidism for many years. Three publications described skeletal abnormalities more than a generation ago. The first report is to be found in the writings of von Recklinghausen,[1] in 1891. He reported the case of a girl of twenty-three who had had severe hyperthyroidism for five years; at necropsy the bones sawed like "rotten wood" and the calvarium and sternum could be cut with a knife. Histologic examination showed that resorption spaces were abundant in the cortex, which often contained nests of osteoclasts. Koeppen,[2] in 1892, reported on the apparent relation between osteomalacia and hyperthyroidism. Again in 1901, Askanazy[3] reported the case of a woman, aged fifty-four, who had suffered for five years from hyperthyroidism due to an adenomatous goiter. Necropsy revealed gross osteoporosis, and microscopically the bones showed extensive osteoclastic resorption.

For some reason there was a long interval before the roentgenologic demonstration of the calcareous demineralization of the bony system in cases of hyperthyroidism was made by Kummer[4] in 1917. Bernhardt,[5] in 1927, reported the same roentgenologic changes in the bones of a woman of forty-seven years who had hyperthyroidism. There was marked decrease in density of the bones, with thinning of the cortex.

Plummer[6] and Dunlap and Moore,[7] in 1929, noted these bone changes in 5 cases of hyperthyroidism, in 4 of which there was exophthalmic goiter and in 1 adenomatous goiter. In 2 cases the bone changes in the posterior ribs were so extensive that the question of metastatic malignancy was considered; in 1 case the diagnosis of malignant degeneration of the thyroid with bony metastasis was ventured. In 1 there were multiple fractures of the ribs at autopsy; they were very friable and could be crushed between the fingers. The ages varied from twenty to sixty-four years, with the duration of disease from one to thirteen years. Those patients in whom the disease was of short duration had high basal metabolic rates. A number of other contributors, including Snapper,[8] Henricke,[9] Gurshman[10] and Hunter,[11] have also described bone decalcification in this disease.

All the bones of the skeleton are involved; the small and flat bones show the change first, and later in the disease it is evident in the spine and long bones. Hunter found that in less than half the cases of exophthalmic goiter the bones were decalcified. In Coryn's[12] patient the degree of bone softening was so severe that the patient decreased in height from 1.65 to 1.39 m. In a bedridden patient of Means, Hertz and Lerman,[13] who had thyrotoxicosis and heart failure, dorsal kyphosis and flaring of the lower ribs occurred, and in another patient, described later in this paper, a

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compression fracture of the first lumbar vertebra was found.

BASIS OF THE BONE CHANGE

A conclusive explanation for the basis of the bone changes in cases of hyperthyroidism was given by Aub and his collaborators[14] in 1929. Even prior to this time a negative calcium balance in patients with hyperthyroidism was suggested as the basis of this decalcification, but adequate proof was lacking. They reported an increase in the excretion of calcium and phosphorus in clinical and therapeutic hyperthyroidism. Whereas a normal individual excretes 12.7 gm. of calcium per kilogram of body weight, the average excretion in cases of exophthalmic goiter was found to be 42 gm. per kilogram, or a 231 per cent increase. No change occurred in the level of the calcium or phosphorus in the blood serum. Hunter reported increased values for the serum phosphatase in cases of hyperthyroidism, but they were not nearly so high as in Paget's disease. This overexcretion of calcium was not attributed to mere elevation of the metabolic rate, since it does not occur in patients with high rates resulting from leukemia and fever. In patients with hyperthyroidism treated by iodine or thyroidectomy, Aub and his co-workers observed a return to normal in the calcium excretion. They considered the most likely cause for this calcium depletion of the bones to be a direct stimulating effect of the secretion of the thyroid gland on the calcium deposits therein. Parathyroid stimulation either directly or by way of the sympathetic nerves was considered but was thought not to be plausible.

Moehlig and Adler[15] believed that these bone changes in cases of hyperthyroidism were secondary to a disturbance in the metabolism of carbohydrates. They attributed the bone changes in Paget's disease to an alteration in glycogen metabolism, probably secondary to changes in the pituitary gland. The bone changes in cases of hyperthyroidism were explained on a similar basis.

Aub and his co-workers found no relation between the level of the basal metabolic rate and the calcium output. That duration of the disease might play a role in the extent of the osteoporosis was suggested by their report of a patient with exophthalmic goiter of seventeen years' duration in whom marked decalcification of the bones was demonstrated. Hunter found no relation between the duration of the disease and the degree of osteoporosis. Means and his co-workers classified the decalcification in cases of hyperthyroidism as a specific type of inanition. The degree or rate of decalcification depends on the duration of the disease, the rate of calcium loss and the calcium intake.

Table 1. *Spontaneous Fractures in Hyperthyroidism.*

AUTHOR	NO. OF CASES	AGE	SEX	DURATION OF HYPERTHYROIDISM yr.	BONES FRACTURED	X-RAY FINDINGS
Hunter, 1930	2	No details given				Decalcification of entire skeleton
Coryn, 1936	1	61	F	9	*a.* Wrist *b.* Radius *c.* Left femur (intertrochanteric) *d.* Left femur (lower third)	Extreme decalcification of entire skeleton
Gottlieb and Schachter, 1937	1	53	F	5	*a.* Right humerus (diaphysis) *b.* Right humerus (supracondylar)	Intense decalcification of entire skeleton Cortex like a thin line
Means, Hertz, Lerman, 1937	1	—	F	3 (fracture) 9 (postoperatively)	1st lumbar vertebra (compressed)	Marked decalcification of spine and pelvis Follow-up x-rays showed no recalcification
Present cases	2	58	F	9	*a.* Right femur (neck) *b.* 1st lumbar vertebra (compressed), 14 mo. later	Scoliosis Marked skeletal decalcification
		78	F	17	*a.* 6th and 10th dorsal vertebrae (compressed) *b.* 8th dorsal vertebra (compressed), 1 yr. later	Marked decalcification

SYMPTOMS

Very little has been reported concerning the subjective effects of these bone changes in cases of hyperthyroidism. Von Recklinghausen's patient, aged twenty-three, who had had exophthalmic goiter for five years, complained of pain in the back and arms with cramp in the feet and hands. Askanazy's patient, aged fifty-four, with adenomatous goiter of five years' duration, suffered severe backache following a fall. No mention was made of the possibility of a fracture of the vertebrae. Snapper stated that the condition produces much generalized pain. Coryn reported a patient, aged thirty-five, who had painful osteoporosis of the tibias. Means spoke of the deep aching pains in

the extremities and back and also of painful ribs. Moehlig and Adler also mentioned the painful bones in this condition.

SPONTANEOUS FRACTURES

A review of the literature revealed only 5 cases of hyperthyroidism in which spontaneous fractures had occurred. In these cases the resulting osteoporosis was accepted as playing a potent role in the production of the fractures. In Table 1 is given a clinical summary of these cases. Hunter did not give the details of his 2 cases, except to mention the occurrence of a spontaneous fracture in a case of exophthalmic goiter in which the roentgenogram demonstrated decalcification not only of the fractured bone but also of all other bones of the skeleton. Coryn's patient had multiple fractures, four in all, and Gottlieb and Schachter's[16] patient, within a period of three months, suffered two fractures of the right humerus, each following a minor injury. Means's report is of especial interest in that his patient had had a thyroidectomy nine years previously for exophthalmic goiter, a condition she had had for more than three years before operation. She suffered a sudden pain in the back, and on roentgenologic examination marked decalcification of the vertebrae and pelvis was noted, with a compression fracture of the first lumbar vertebra. In this case calcium and viosterol were administered, with relief of symptoms, but roentgenograms taken later did not give evidence of recalcification.

CASE REPORTS

Case 1. This patient was first seen at the clinic in 1928, when she was 50 years of age. Her symptoms were easy fatigue, palpitation, nervousness and enlargement of the thyroid gland of 1 year's duration. She weighed 103 lb., having lost 4 lb. The basal metabolic rate on two determinations was +35 and +27 per cent. A diagnosis of primary hyperthyroidism was made, and after the usual period of administration of iodine and rest in bed a subtotal thyroidectomy was done. A moderately severe reaction occurred postoperatively and a normal convalescence ensued. A little more than 1 year later a second thyroidectomy was necessary for recurrent hyperthyroidism. Following this it was necessary for the patient to take iodine in an attempt to control the symptoms of recurrent hyperthyroidism. She did not return for observation between September, 1932, and December, 1936, but when seen on the latter date she was obviously hyperthyroid; she weighed only 94 lb. and auricular fibrillation was present. She had noticed progressive heat intolerance, excessive perspiration and a gradual loss of 20 lb. in 3 years. In August, 1935, she tripped on a doorstep and fell on her right hip, fracturing the right femur. The fracture healed promptly, but while she was confined to bed for this condition she began to have continuous aching pain in the back. After being up and around, she noticed that her back was quite rounded. Five months later she began to suffer pain in the lower part of the back, especially on the left side. This pain was periodic but was growing gradually worse.

Table 2. *Clinical course of Case 1.*

DATE	BASAL METABOLIC RATE	PULSE	WEIGHT	CONDITION	REMARKS
			lb.		
2- 2-28	+27		103	Hyperthyroid	Subtotal thyroidectomy
5-18-28	+23	104	112	Good	Advised to take iodine
2-18-29	+35	112	109	Hyperthyroid	Advised to continue iodine
3-11-29					Subtotal thyroidectomy
1-14-30	+18	92	118	Good	
4-10-30	+20	92	118	Slightly hyperthyroid	Advised to take iodine
9-17-30	+10	92	113	Good	Advised to continue iodine
9-16-31	+ 8	96	120	Good	Advised to continue iodine
9-16-32	+18	100	110	Slightly hyperthyroid	Advised to continue iodine
8- ?-35					Fractured neck of right femur
12-16-36	+39	100*	94	Hyperthyroid	Thyroidectomy advised
12-26-36					Subtotal thyroidectomy
7-27-37	+13	84	97	Good	Advised to take iodine
1-31-38	+ 2	100	107	Good	Discontinued iodine

*Auricular fibrillation.

Examination of the back revealed some rigidity of the spinal column, especially in the lumbar region. Forced flexion of the spinal column caused pain in the lower portion. There was moderate kyphosis and scoliosis. Roentgenologic examination revealed scoliosis of the lumbar portion of the spinal column, with marked decalcification of the lower thoracic and lumbar vertebrae, sacrum, pelvis and skull. The first lumbar vertebra was crushed (Fig. 1).

The basal metabolic rate was +39, which was higher than at any previous time. The value for the hemoglobin was 72 per cent; erythrocytes numbered 3,830,000. The values for the serum calcium and phosphorus were 8.8 and 3.7 mg. per cent, respectively, the serum phosphatase being 2.57 Bodanski units. Examination of the urine gave essentially negative results.

On December 26, 1936, a subtotal thyroidectomy was done, remnants of both lobes and the isthmus being removed. The pathologist's diagnosis was primary hyperplasia of the thyroid gland. A moderately severe postoperative reaction occurred, but the patient then went on to a normal convalescence. Orthopedic management, to be described below, was instituted shortly after the operation when the patient's condition warranted. Improvement was obtained in the back pain by these measures, and when she was seen a little over a year later (January 31, 1938), her general condition was good, the basal metabolic rate was +2, she had gained 10 lb., and there were no symptoms referable to the back.

Case 2. This woman, 78 years old, was first seen at the clinic in November, 1937. She had been perfectly well until the age of 61, when she became nervous, had palpitation and lost weight, heat intolerance developed, and she noticed that her eyes were becoming prominent. Five years after the onset of these symptoms a diagnosis of hyperthyroidism was made and a subtotal thyroidectomy was done elsewhere. After the operation she improved somewhat from the standpoint of her cardiac symptoms, but never regained her usual health. She continued to lose weight and was intolerant to heat. At times she had

Figure 1. *Case 1, showing crushed lumbar vertebra.*

a recurrence of cardiac symptoms, which could be partially controlled by taking iodine; this she continued to do periodically up to the time of admission to the clinic.

At the age of 76, 2 years before coming to the clinic, the patient began to notice increasing rounding of the back with shortening in stature. With these changes she had diffuse back pain and cramps in the legs. One and a half years before admission she suffered an injury to her back by a minor fall. A roentgenogram taken at that time revealed decalcification of the bones of the spinal column with a compression fracture of the sixth and tenth dorsal vertebrae. She was fitted with a brace, with relief of pain.

Again 3 months before admission, while stooping over before putting on her brace, she suffered a recurrence of pain in the back, and a roentgenogram taken at that time revealed that the eighth dorsal vertebra was fractured. The question of hyperparathyroidism was considered and the patient was referred to the clinic for study.

Physical examination revealed a frail, alert, elderly woman with moderate exophthalmos. She weighed 79 lb.; the pulse rate was 92. She had a fine tremor, the body was warm and moist, and the precordial impulse was forceful. There was a slight to moderate pitting edema of the legs. Examination revealed thyroid remnants on the right and left, measuring 3 by 4 cm., with a bruit over the right superior pole.

The basal metabolic rates were +21 and +33. Examination of the urine gave negative results. The hemoglobin was 69 per cent; erythrocytes numbered 4,510,000 and leukocytes 5950. The value for the nonprotein nitrogen

Figure 2. *Case 2, showing multiple crushed vertebrae.*

was 26 mg. per cent, the uric acid 2.9 mg. per cent, and the phosphorus 4.8 mg. per cent. The serum phosphatase was somewhat elevated, being 6.7 and 7.3 Bodanski units on two occasions. Roentgenologic studies revealed a decreased density in all the bones, with a very thin cortex. There was marked mid-dorsal kyphosis, with narrowing of the mid-dorsal bodies, especially the sixth, eighth and tenth vertebrae (Fig. 2). The patient received the usual preoperative treatment and underwent a subtotal thyroidectomy without incident. The specimen showed primary hyperplasia.

ORTHOPEDIC TREATMENT

The outstanding complaint of these patients requiring orthopedic treatment was the generalized back pain due to kyphosis and the associated collapse and crushing down of various vertebral bodies. In both cases crushing of the ver-

tebrae had occurred at relatively long intervals before admission to the clinic, so that actual correction of the deformity could not be expected as in an acute fracture of the vertebral body.

The most effective immediate therapy to relieve the back pain is progressive extension of the spinal column. The term "progressive" is used advisedly because it has not been possible to place these patients immediately in a supine position on a firm fracture bed. The consequent discomfort is so great that they will not tolerate the position, but they will do so if it is approached by easy stages.

This treatment is best instituted about a week to ten days following the thyroidectomy, because in the immediate postoperative period the patient is much more comfortable if supported in a semisitting position in bed. Once the acute postoperative symptoms have subsided, however, the patient is gradually lowered into the supine position. This is carried out gradually in an attempt at actual hyperextension of the spinal column, provided the patient will tolerate the maneuver.

This procedure, combined with intensive physiotherapy in the form of hot fomentations and dry heat from an infrared lamp and massage of the back, will usually relieve the back pain. The patient is then maintained in the maximal position of hyperextension that can be tolerated. The next step is advocated in order that the individual may sooner become ambulatory. At this stage a plaster jacket is applied, intravenous Pentothal anesthesia being utilized for the patient's comfort. Following this, the patient is allowed to be up and about and then returns home. The cast is well tolerated. It is worn for a minimal period of six to eight weeks, after which a high-type Taylor back brace, with a wide abdominal apron or preferably a corset in which the brace is incorporated, is worn. In some individuals plaster body casts are not accepted. Such patients are kept in bed longer and finally mobilized with the aid of the corset brace already described.

The further general line of treatment is obviously that of assisting the patient in every way possible so that the bones may recalcify. Calcium gluconate, to the amount of 60 gr. daily, and a high vitamin intake with a well-balanced average diet have been advised.

COMMENT

The osteoporosis of hyperthyroidism has not attracted great interest because it is usually asymptomatic. The occurrence of these extensive bone changes is additional evidence of the metabolic disturbance and widespread alteration of body physiology in this disease. The apparent reason for the usual absence of symptoms referable to bone in cases of hyperthyroidism is that the disease must of necessity be of long duration to produce significant effects. Usually the patients with hyperthyroidism come for treatment in the first year or two of the development of the disease, before very marked bone changes have taken place. In those cases reported in the literature in which these bone changes had occurred the disease usually had been present longer than five years; in the 2 cases here reported it was present for nine and seventeen years, respectively. Usually the patients who have this condition have had a low grade of hyperthyroidism which has been permitted to continue for years before a diagnosis has been made or treatment instituted.

The teeth of hyperthyroid patients have, as a rule, a tendency to early caries and alveolar absorption. It is conceivable that the general decalcifying process has in some cases extended to the tooth structures, producing lowered resistance to caries. Contrary to this observation and assumption is the view of Schour,[17] who believes that present evidence has not established a correlation between calcium metabolism and caries.

In the first case here reported, roentgenograms were taken one year after the last operation; no recognizable bone recalcification was found. The patient, however, has since observed marked lessening in the back pain. Moehlig and Adler did not believe that benefit could be derived from thyroidectomy. They reported a patient with hyperthyroidism due to an adenomatous goiter in whom the osteoporosis of the vertebrae and pelvis progressed in spite of a thyroidectomy. In this case calcium was deposited in the spleen. Means's patient after the thyroid was removed showed improvement in the kyphosis and flaring of the ribs. Some of the improvement was attributed to an adequate diet. It is obvious that if a patient has a negative daily calcium balance to the extent reported by Aub, and if this continues for a number of years as it did in most of these cases, it will take considerable time to replace this loss with an average individual daily calcium intake of a little more than 1 gm. a day. This should explain the failure of recalcification to be apparent on roentgenologic examination one year after operation in the case here reported. However, in Means's cases the fracture did not take place until nine years after the thyroidectomy was performed, which tends to indicate that recalcification does not occur. In the second case here reported, the patient of seventy-eight years, it is accepted that some osteoporosis may have occurred on the basis of senile change. However, the combination of the senile change and hyperthyroidism produced a more extensive osteoporotic change.

SUMMARY

A review of the literature has revealed 5 cases of single and multiple spontaneous fractures on the basis of the decalcifying process in hyperthyroidism. To this group 2 more cases are added. Both patients had hyperthyroidism of long duration, nine and seventeen years, respectively, and both had suffered multiple fractures.

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ROCKY MOUNTAIN SPOTTED FEVER IN MASSACHUSETTS

Report of a Case

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SINCE the clinical picture of Rocky Mountain spotted fever was first reported by Maxey[1] in 1899 an increasing number of cases of this disease have been recognized in various parts of the country. The first cases in the East were reported in 1925 and 1926 in New York and Virginia. Each year cases of this disease have been discovered in new areas where it had been previously thought not to exist, but until the past summer no cases had been recognized in the New England area. In September, 1937, McLaughlin and Grover[2] reported 2 cases treated in Rhode Island during July, 1937, in which the infection was received in southern Massachusetts in the Cape area. In the case here reported the patient was also infected in southern Massachusetts during July, 1937, and so far as we know this is the first case to be diagnosed and treated in Massachusetts. In the Middle and South Atlantic States, however, where the disease has been recognized for several years, well over 100 cases are being reported each year, proving that the disease is far from being a medical rarity in the East.

From our knowledge of the etiology of Rocky Mountain spotted fever there is no reason why it should not be found more often in Massachusetts. The eastern type of the disease is transmitted chiefly by the dog tick, which is prevalent in Massachusetts, particularly along the South Shore and the Cape, during the summer months. Reports in the literature describing the experiences of physicians in other states[3, 4, 5] stress the fact that before it was generally realized that Rocky Mountain spotted fever could occur in these states many cases went unrecognized. For example, in Maryland up to 1931 no cases of the disease had been reported. In 1931, 38 cases were reported, and since then there have always been over 30 cases a year. Similar experiences are reported in Virginia, North Carolina and the District of Columbia. It would therefore seem quite likely that if the disease is looked for it may be found to be more prevalent than suspected in Massachusetts.

The clinical features of the eastern type of Rocky Mountain spotted fever have been best described by Dyer and his associates,[5, 6, 8] who first urged physicians to be on the lookout for this disease in the eastern states. The best recent summary appeared in the report of a case by Carey and Duncan.[7]

The disease is caused by the bite of infected ticks, which it is believed may inherit the infection from one generation to the next. In the East the disease is transmitted chiefly by the eastern dog tick or *Dermacentor variabilis*. These ticks are prevalent in Massachusetts from March to September, and are most active during June and July. The incidence of the disease is therefore definitely seasonal.

There is an incubation period of two to twelve days, with some vague prodromal listlessness or headache. The actual onset is abrupt, with fever, mild chills, headache, nausea and marked lassitude and prostration. The fever is intermittent in character and gradually increases in severity up to the end of the second week, then terminates

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by lysis during the course of the third week. The prostration and other toxic symptoms parallel the fever in intensity. The pulse is moderately rapid. There is usually a moderate leukocytosis and some albuminuria.

On the second to fifth day after onset the characteristic rash appears. This is maculopapular and rose-red, and varies in intensity with the height of the temperature. It becomes deeper and more extensive, tending to become purplish and in some cases confluent, during the second week. With the beginning of lysis the rash begins to fade and becomes brownish, lasting altogether about three weeks. The initial location and the spread and final distribution are important, especially in differentiating typhus fever. In Rocky Mountain spotted fever the rash appears first on the wrists and ankles, and spreads rapidly during the first two days to the back, arms, legs and chest, and finally to the abdomen. Occasionally there are spots on the face and scalp. Lesions on the palms and soles are frequent. In typhus fever the rash starts on the chest and abdomen and may or may not spread to the extremities. The typhus rash also appears later, is usually milder, and often disappears before the end of the second week.

The pathognomonic feature of Rocky Mountain spotted fever is the production of a positive Weil-Felix test, that is, a positive agglutination response to one or more strains of *Bacillus proteus* X. Usually agglutination is found for *B. proteus* X-19 in a titre 1:160 or more and may reach as high as 1:10,000. In Rocky Mountain spotted fever this agglutination reaction typically occurs late in the disease, may be negative for the best part of the first two weeks (in distinction to typhus, where it usually is positive during the first few days), and may be quite weak until late in convalescence. In suspicious cases, therefore, repeated Weil-Felix tests should be done up to the end of the fourth week or later, even though the patient may have been clinically well long before that time. Agglutinins are sometimes also found for *B. proteus* X-2, which may serve as a differential point from typhus fever in which no agglutinins for X-2 are found. Occasionally there are agglutinins for X-2, without appreciable agglutinins for X-19. This fact is of some practical importance since in many laboratories when a routine Weil-Felix is done only agglutination for *B. proteus* X-19 is carried out.

Table 1. *Clinical Features of Rocky Mountain Spotted Fever (Eastern Type) and Typhus Fever Compared with Case Reported.*

Clinical Feature	Rocky Mountain Spotted Fever, Eastern Type	Typhus Fever	Case Reported
Incubation period	2-12 days	6-14 days	2 days
Initial symptoms	Chills, fever, headache, anorexia, prostration.	Chills, fever, headache, anorexia, prostration.	Chills, fever, headache, anorexia, prostration.
Later signs	Hyperesthesia, rash. May have rigid neck, stupor or delirium, cough.	Hyperesthesia, rash. Cough and delirium usual, more severe.	Hyperesthesia, rash, rigid neck, semi-stupor; no cough.
Rash:			
Character	Maculopapular	Maculopapular	Maculopapular
Onset	2nd to 5th day	5th to 7th day	4th day
Starts	Forearms, legs.	Chest, abdomen	Forearms, legs.
Spreads	Chest, abdomen.	Occasionally to extremities	Chest, abdomen.
Duration	Fades slowly end of 2nd week, lasting through 3rd week.	May last only few days, usually gone by end of 2nd week.	Started to fade 11th day, gone 21st day.
Temperature	Rarely over 104°F. Daily remissions 1 to 3 degrees. Gradually increases first few days.	Usually 104°F. or over; less marked remissions. Sudden rise to high temperature at onset.	P. M. temperature 103° to 104°F.; A. M. 100 to 101°F. Highest temperature gradually rose from 101 to 103.2°F. in 4 days.
Duration of fever	Slow lysis, 12th to 22nd day.	Rapid lysis, 14th to 16th day.	Slow lysis, 14th to 20th day.
Pulse	Relatively slow (around 100)	Higher in proportion to temperature.	Averaged 90 to 115
White blood cells	Normal or slight leukocytosis	Usually normal.	5400-12,000 (21,000 after liver extract)
Urine	Albumin, casts	Albumin, casts	Albumin, casts
Weil-Felix Test	Negative 1st week, positive 2nd week, highest end of 4th week.	Positive 4th to 7th day, highest end of 2nd week.	Negative 6th day, positive 1:80 13th day and 18th day.
Mortality	25% (western type, 80-90%)	5-70%	

These clinical features are very striking and characteristic, and once the diagnosis is thought of there is usually little difficulty in confirming it. Typhus fever is quite similar, but as mentioned above there are important epidemiologic, clinical and laboratory differences. These differences are emphasized in Table 1, in which the chief features of eastern Rocky Mountain spotted fever have been summarized and compared with the comparable features in typhus fever and with the signs and symptoms noted in the case reported below.

There is no accepted specific treatment for Rocky Mountain spotted fever. There is a prophylactic

vaccine available prepared from infected ticks, which may be given in two injections and is reported as being effective. The most important preventive measure has been to search the body for the presence of ticks every eight to twelve hours, since the virus is not activated and cannot be transmitted until the tick has fed for several hours. In regions where infected ticks are known to be present, examination for ticks every twelve hours has been established as a routine in most summer camps. There is some slight evidence that natives are not so susceptible to the tick infection as are nonresidents. In this connection it is interesting to note that in the 2 Rhode Island cases and our case all the patients were tourists from outside the State.

CASE REPORT

Mrs. L. W., a 29-year-old, white woman (Case C4904), was admitted to the Newton Hospital, July 27, 1937, complaining of headache and fever of undetermined cause. She first felt ill 5 days before entrance to the hospital. She had a slight headache in the morning. That evening she had a chill and was nauseated, the temperature rose to over 101°F., and she felt headache and grippy pains over the body. The next morning she continued to have a temperature and headache and seemed lethargic. Physical examination showed a red throat and slight resistance on flexing the neck. The patient was dizzy when sitting up. The temperature was 102°F. During the next 3 days she continued to run a temperature, varying between 103° and 99.5°F. She became drowsier and complained of continued headache, dizziness and nausea. There were no specific complaints referable to the throat, chest or abdomen. Physical examinations continued to be essentially negative except for some tenderness and resistance on flexing the neck and very feeble knee jerks. Urine examinations were negative.

On the day of admission a scattered, pink, maculopapular rash was noticed for the first time, on the forearms and legs, but not elsewhere. The patient continued to have headache and lethargy, which did not improve, and was sent to the hospital for further study. Later questioning for a possible contact with a virus disease revealed the information that about 10 days previously she had started traveling from Indiana by automobile, stopping one night in Ohio and spending one or two nights in Washington, District of Columbia. Forty-eight hours before she became sick she noticed a swollen area on the back of her right hand with a definite puncture opening in the center, which she thought was an insect bite. The swelling disappeared during the day and left no trace. At this time she was visiting in the town of Egypt, Massachusetts, and had just spent the day driving down the Cape to Provincetown and back. The only drugs she had had before the appearance of the rash were a few doses of Luminal, Emagrin and aspirin, all of which she had taken on former occasions without reaction, and one injection of morphia and atropine 2 days before the rash occurred.

The patient had had several attacks of tonsillitis during the past year: this was the only comment on her past history by her family physician in Indiana. She was supposed to have had measles three or four times.

Physical examination on admission showed a well-developed and nourished woman who appeared sick and toxic. She was lethargic. It was difficult to discover her symptoms except for some headache, and dizziness on sitting up. The skin was pale. There was a maculopapular rash on the arms and legs, chiefly on the extensor surface. The macules averaged 3 to 6 mm. in diameter, were pinkish, and faded on pressure. They were more prominent when the temperature was high. There was no rash on the face or body. The pupils reacted normally and were equal and regular. The fundi were normal. The ears and nose were negative, and there was no tenderness over the sinuses. The throat was quite red but there was no mucus or fibrin. The tongue was heavily coated. No ulcers or rash were seen in the mouth or palate. The cervical glands were not enlarged. The thyroid was not enlarged. The heart was normal in size, regular and without murmurs. The pulse rate was 96 and the blood pressure 120/80. The lungs were negative. The breasts were normal. The abdomen showed no tenderness or masses. The spleen and liver were not palpable or tender. The kidneys were not palpable. There was no tenderness over the spine. There was definite resistance to flexion of the neck, and this appeared to cause pain. There was a suggestion of a slight Kernig's sign. However, the knee jerks were almost absent and could not be obtained without re-enforcement. Babinski's reflex was negative. The arm reflexes were weak. A more detailed neurologic examination was unsatisfactory because of lack of co-operation.

On admission the urine showed a specific gravity of 1.023; the sediment showed a slight trace of albumin, no sugar, 2 to 5 white cells per high-power field and a rare hyaline cast. The albumin and white cells were constantly present during her stay in the hospital, the former increasing slightly at the height of the temperature. The nonprotein nitrogen of the blood was 40 mg. per cent on admission and 29 mg. per cent 3 days later. Frequent blood counts were done. On admission the white-blood-cell count was 5400, with 22 per cent band cells, 54 per cent segmented polymorphonuclears and 24 per cent lymphocytes. The white-cell count gradually increased to 17,000 on the 4th day after admission and to 21,000 at the height of fever a week after admission, thereafter dropping gradually to a normal of 9800 the 2nd week after admission. The differential rose to 56 per cent band cells and 22 segmented polymorphonuclears on the 2nd day and then averaged 35 to 40 and 40 to 45 per cent respectively for the next 3 days, after which it gradually returned to 18 to 48 per cent respectively. The red-cell count was 4,900,000 and the hemoglobin 97 per cent on admission. There was a gradual decrease to 3,700,000 red cells with a hemoglobin of 68 per cent on the 5th day and to 3,350,000 red cells with a hemoglobin of 69 per cent on the 8th day. Following two transfusions, the final count was 3,830,000 red cells with a hemoglobin of 77 per cent. A blood icteric index taken on the 10th day was 15. Five blood cultures were taken at intervals of 24 to 48 hours and planted on differential media, but showed no growth. A throat culture on admission showed the presence of a hemolytic streptococcus. Stool cultures were negative. A blood Hinton test was negative.

A lumbar puncture was done the day after admission and showed clear fluid with an initial pressure of 130 mm. of water. The cell count was 10 per cubic millimeter and the sugar content 54 mg. per cent. Albumin and globulin were negative. Stained smears showed no organisms. A second lumbar puncture done 6 days after admission showed clear fluid and an initial pressure of 170 mm. The dynamics were normal. The cell count showed 32 white blood cells per cubic millimeter. The

albumin and globulin showed no increase. The sugar content was 83 mg. per cent. A culture was negative.

On the day after admission, the 6th day of the disease, a blood Widal test was negative. Agglutination tests with *Salmonella paratyphosus* B and *Brucella abortus* were negative, as was a Weil-Felix test. One week later the Weil-Felix test was positive at a serum dilution of 1:80 and negative at 1:160. There was a partial Widal agglutination; that against *S. paratyphosus* B was negative. Five days later the Weil-Felix reaction was again positive at 1:80 and negative at 1:160.

X-rays of the heart and lungs were taken on the 2nd and 5th days after admission and were entirely negative.

For 7 days after admission she ran an irregular temperature ranging from 100° to 104°F., with her pulse rate ranging from 90 to 120. The respirations remained at 20. She had one or two slight chills each day, following which she would usually complain of headache, which sometimes required morphia for its relief. The state of lethargy remained unchanged. She was obviously toxic. The moderate rigidity of the neck continued unchanged. She showed no other change in physical examination except in the character of the rash, which during the first 48 hours spread upward over the arms and legs to cover the abdomen and chest, with a few spots on the face. In many areas the papules became confluent over the arms and legs. Four or 5 days after onset the color of the rash became more purplish and some of the papules did not fade completely on pressure. During the next few days the rash became definitely purplish, but no definite petechiae or hemorrhages were ever seen. On the 7th day after its appearance the rash began to fade, disappearing first on the chest and abdomen and on the flexor surfaces of the arms and legs.

Shortly after this the patient's temperature began to fall and general improvement was noted. The rash remained quite prominent for another week, gradually fading out and disappearing finally from the arms and legs. On the 9th day after admission the temperature dropped to a maximum of 102°F., and fell gradually to a maximum of 99°F. on the 14th day and 98.6°F. on the 16th day, this being approximately the 20th day of the disease. The pulse returned to normal gradually during the same period.

Coincident with this drop in temperature and pulse, the lethargy, headache and stiff neck cleared rapidly. The patient's appetite returned. Except for weakness and slight slowness of reaction, she seemed entirely well.

Because of the hemolytic streptococcus which was isolated from the throat, sulfanilamide therapy was instituted the day after admission, 80 gr. being given the first 24 hours and 5 gr. every 3 hours for the next 4 days. This appeared to have absolutely no effect except possibly to contribute to the development of a moderate secondary anemia.

Because of the leukopenia and marked decrease in the number of segmented polymorphonuclear cells, with increase in the number of bands, a series of intramuscular liver extract injections were given, 1 cc. (15 units) every 8 hours for the first 3 days. During this administration the white blood-cell count, which was taken every 12 hours, steadily increased to 17,000. Thirty-six hours after administration of liver extract was stopped the white-cell count dropped to 12,000, with a coincident drop in the percentage of band cells from 35 to 23. Daily liver extract injections were resumed for the next 3 days, during which time the white-cell count stayed at 21,000. When the liver extract was again stopped the white-cell count again fell to 14,000, but this decrease was in accordance with the beginning clinical improvement of the patient.

Two blood transfusions were given, with the patient's husband as donor. These were advised because of the anemia rather than because of any anticipated specific effect. The first was administered 1 week after admission, 450 cc. of citrated blood being supplied. The next day the patient's maximum temperature was lower than it had been for a week, and it continued to go down steadily. A second transfusion was given 3 days later because of the progression of the anemia. Following this transfusion the temperature continued to fall, and it was never above 101°F. thereafter. The anemia was controlled. The effect of the transfusion may well have been coincident with the natural termination of the disease, the first transfusion being given on the 12th day and the second on the 15th day of the disease.

Other treatment was symptomatic. Intravenous injections of 1000 cc. of 10 per cent glucose were given on several occasions when the fluid intake was inadequate. Aspirin 10 gr. was given occasionally for the headache, although 1/6 gr. of Pantopon was found more satisfactory.

The patient was discharged on the 20th day after admission, the 25th day after the onset of the disease, feeling quite well. She returned to her home in Indiana 2 weeks later.

Follow-up reports from the patient, the last in July, 1938, stated that she had been examined at intervals by her family physician and continued in good health. Her tonsils were removed at his advice in November, 1937, since he believed that they had long been a source of chronic infection.

COMMENT

The important etiologic feature of this case is that although the patient lived in the Middle West, the infection was apparently acquired in Massachusetts. The season of the year, early summer, was the time when the dog ticks which transmit the infection are known to be particularly active.

The chief difficulty in this case lay in making the diagnosis. The possibility of Rocky Mountain spotted fever did not occur to us for several days, and when we first considered it we thought it was a rather far-fetched possibility. After reading descriptions of eastern cases of the disease, however, the similarity to our case was so obvious that we had little hesitancy in making the diagnosis even before a report of a positive Weil-Felix reaction was returned. Unfortunately, not being familiar with the serology of this disease, we did not appreciate the importance of obtaining further Weil-Felix tests late in the third and fourth weeks of the disease after the patient was clinically well.

From the point of view of differential diagnosis it may be of interest to review the diagnoses which we considered before the possibility of Rocky Mountain spotted fever was suggested. The onset of the disease resembled the grippe type of infection. This diagnosis was soon abandoned. The possibility of pyelitis, typhoid or a centrally located

bronchopneumonia was next considered. The development of mild meningitic symptoms suggested the possibility of encephalitis, atypical meningitis or a brain abscess. The early lumbar-puncture findings were against these last possibilities, beyond indicating the presence of a mild encephalitis which would hardly have been enough by itself to account for the severe prostration and toxemia. When the rash first appeared typhoid fever or a meningococcus or streptococcus septicemia (especially of a subacute bacterial endocarditis) were strongly considered, although the rash was not typical of any of them. As noted above, five blood cultures taken at frequent intervals were negative, as were stool cultures and blood tests for typhoid, paratyphoid and undulant fever. The presence of a hemolytic streptococcus in throat culture was given some significance, especially in connection with the leukopenia. Sulfanilimide therapy was tried, with no effect whatsoever. The blood smear ruled out agranulocytic angina, but in view of Murphy's success with liver extract in combating the leukopenia of this disease, large doses of liver extract were given. Haverhill fever was considered, but joint symptoms were lacking. The negative Weil-Felix reaction on the sixth day and the character of the rash seemed to eliminate typhus fever. The result of all this laboratory investigation was that at the end of a week we had a patient who was extremely, perhaps fatally, ill, yet we had no satisfactory diagnosis. At this point the resident house officer, Dr. Mario C. Caturani, who was interested in the case although it was not under his direct care, suggested the possibility of Rocky Mountain spotted fever, pointing out that the clinical picture was quite typical of this disease. This diagnosis was confirmed by the further progress of the case and the later development of a positive Weil-Felix reaction.

Little can be said about treatment of this patient except that it was necessarily chiefly symptomatic. The liver-extract injections appeared to favor the development of a leukocytosis with a fair percentage of adult polymorphonuclear cells, which we felt might have some value in combating the infection. The two blood transfusions definitely improved the general condition of the patient.

SUMMARY

A case of Rocky Mountain spotted fever, eastern type, which occurred in Massachusetts in July, 1937, is reported. Two other cases which received their infection in Massachusetts during July, 1937, but were treated in Rhode Island have also been recently reported. These cases are apparently the first instances of this disease to be diagnosed in the New England area.

From the experience in other states it is probable that as soon as it is generally realized that Rocky Mountain spotted fever can occur in this section of the country more cases will be diagnosed. The importance of recognizing the occurrence of this disease is, first, that it carries a mortality comparable to lobar pneumonia, and secondly, that by proper preventive measures it should be possible to control the spread of the disease. These measures should include steps to eliminate ticks in the infected areas, frequent routine examinations of the body for ticks (every eight to twelve hours) by persons exposed to them during the summer months and the possible use of prophylactic vaccine.

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THE INGESTION OF IODINE AS A METHOD OF ATTEMPTED SUICIDE

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THE experience of being called to attend a patient who has ingested iodine in a suicidal attempt is not uncommon among physicians engaged in general practice, and is a frequent occurrence in hospitals. Too often this situation is treated purely as a problem in internal medicine. The basic factors which have motivated the act are not given proper weight, owing in part to lack of understanding on the part of physician and family that suicide represents the patient's solution of deep psychological drives, normally resolved in other modes. A suicidal attempt may be a hasty act — the result of a sudden conviction — or it may have been planned for some time. In the latter case it is more often successful. Usually the act is not caused by a single factor, and it is rarely the first symptom of the mental disturbance which it follows.

THE RARITY OF FATALITIES FROM IODINE POISONING

In the course of a further study of 1195 cases of attempted suicide treated at the Boston City Hospital between 1915 and 1936[1] it was learned that 327 individuals† sought to end their lives by ingesting iodine in some form. Usually the tincture was chosen, possibly because of its ready availability, but iodoform and other forms such as Lugol's solution and iodides were also used. None of these patients were successful in terminating life by this means, and in the single fatal case in which iodine figured (a man of sixty who had taken a considerable amount of bichloride of mercury at the same time) death was due to the effects of another toxic agent rather than to iodine.

Oddly enough, although iodine is used in suicidal attempts more often than any other poison, fatal cases are very rare. The Medical Examiner for Suffolk County, Dr. Timothy Leary,[2] states that he has never seen a case of fatal iodine poisoning in spite of long experience in investigating sudden and violent deaths in the area served by the Boston City Hospital. Since all such cases occurring in the hospital or without are brought to his attention, the conclusion is warranted that no fatal cases have occurred in this district during the period covered by this study. A few cases have been reported in the medical literature, but these are rare in comparison with the number of attempts which are made.

Canavan,[3] in a long pathological experience dealing with institutional cases, has seen 1 fatal case occurring in a woman of seventy who first attempted suicide by drinking floor polish; later she drank about 120 cc. of tincture of iodine and expired in fifty-five minutes. Postmortem examination revealed an edema of the glottis and extensive necrosis of the mucosa of the stomach. The kidneys were markedly injected. Death was certified to be due to "ingestion of tincture of iodine, suicidal," but there may have been other contributory factors. Peterson, Haines and Webster[4] cite a case described by Herrmann[5] in 1869 which was successful. A man, aged thirty-eight, drank 60 cc. of tincture of iodine and died after thirty-three hours. They also cite the case referred to by Bellot[6] in 1893 of recovery in four days of a woman who drank 120 cc. of tincture of iodine. Culpeper[7] reported a case of a negro boy who died three and a quarter days after his body had been painted with tincture of iodine over one-third its area.

Recently a study has been published by Finkelstein and Jacobi[8] of 18 fatal cases coming to the attention of the medical examiner's office in New York City. They state that up to 1911 only 31 fatal cases had been reported, and that from 1912 to 1935, of 1838 cases of poisoning treated in Berlin, Germany, only 1 was due to iodine. These authors believe that such deaths are due to an unusually large dose or to delay in calling help. Between 1931 and 1937 there were 18 successful suicides by iodine in New York City. Of this group, 13 were men and 5 women. Death in all cases occurred within forty-eight hours, and in 2 cases in one-half and one hour respectively. The dose ingested varied between 1 and 8 ounces. Included in their group was the only case of esophageal stenosis in the literature, occurring after fifty-two days. All cases came to autopsy, and the chief pathologic changes were pulmonary edema, pneumonia (aspirational) and cardiac failure. There were liver changes in 14 cases, but the symptomatology was chiefly confined to the gastrointestinal tract.

The outstanding reason why iodine is so seldom fatal is related to the intrinsic properties of the

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†Since the original study was made and partly as a result of it, a few additional cases of attempted suicide have been found.

drug, for although it is a powerful and slow-acting cell poison, it cannot be absorbed unchanged. It combines with starches, proteins and unsaturated fatty acids or is changed into an iodide before absorption, all of which renders the substance less toxic. The minimum lethal dose has been variously reported between 20 and 150 gr. Iodine is so irritant to the gastric mucosa that the poison is vomited reflexly. Sublethal doses produce symptoms as distressing as fatal doses, and it is usually difficult to ascertain the exact amount that has been swallowed.

The chief and commonest symptoms following iodine ingestion are those of powerful irritation of the gastrointestinal tract, vomiting, purging, extreme thirst, pain in the mouth and stomach, faintness and collapse. Death when it occurs is usually delayed and is due to overstimulation of the thyroid gland (similar to hyperthyroidism), as is also the case in chronic iodine poisoning. There is a secondary effect on the heart, but this is seldom seen in the acute type of poisoning. Iodine is excreted by the kidney, tears, saliva and secretions of the respiratory passages.[9, 10] There is usually some difficulty in swallowing a lethal dose, since the response of the gastrointestinal tract is immediate.

Fatal cases can be viewed with some reservations, since unless there is a cardiac or thyroid factor which sensitizes the patient to iodine, death is commonly due to other causes. From the standpoint of toxicology, iodine is not an important poison. The medical treatment of iodine poisoning should aim to replace the extrarenal loss of electrolytes and water, since there is invariably renal suppression, and nitrogen retention. Forced diuresis as with glucose should be scrupulously avoided.

SUICIDAL CASES OF IODINE POISONING AT THE BOSTON CITY HOSPITAL

In the series of cases studied at the Boston City Hospital, there is some evidence that many cases may have been dramatic gestures and not bona fide efforts at self-destruction. Whether persons who drink iodine are familiar with the fact that it seldom causes a fatality is not known, but it is known that in some instances a sublethal dose is purposely taken. In either case the attempted suicide represents the outcome of intense psychological maladjustment and frustration. In both those patients who are merely being histrionic and those who are actively and actually suicidal, there are definite neurotic drives and often obviously infantile modes of reaction to common situations. In both there is the inability to face reality, or if it can be faced, the unwillingness to seek an adult solution of personal problems. In this sense, the behavior pattern of attempted suicide is closely akin to that of the alcoholic, for in both there is a strong motive of flight.

A number of cases of iodine poisoning at the Boston City Hospital have not been included in this study, for if the hospitalization seemed to be obviously due to accidental poisoning or to an overdose of iodine taken for therapeutic purposes (as with Lugol's solution) the case was not considered eligible for the series. Needless to say, many patients probably claimed that their plight was due to accidental substitution of iodine for other medicines after they had suffered remorse or been frightened and realized what they thought would be the possible consequences of their act. Commonly, friends and relatives conspire to conceal a suicide, and frequently a successful suicide manages to conceal the motive by setting the stage so that his act appears accidental and unintentional. When a psychiatric examination is made of persons suffering from apparently accidental poisoning, it is seen that the mistake may be the result of definite suicidal drives, concealed even from the patient but easily apparent to an examiner acquainted with the force with which the subconscious may order the behavior of an individual. Aggression which normally is directed toward external objects becomes directed to the person, and his life may become the object of his destructive tendencies.

Table 1. *Annual Admissions for Attempted Suicide at the Boston City Hospital, 1915-1936.*

YEAR	IODINE ADMISSIONS	TOTAL SUICIDE ADMISSIONS	PERCENTAGE
1915	0	24	0
1916	3	36	8
1917	5	30	17
1918	2	42	7
1919	4	24	17
1920	1	26	4
1921	7	38	18
1922	14	60	23
1923	20	58	34
1924	11	32	34
1925	21	82	26
1926	28	83	34
1927	21	58	36
1928	15	48	31
1929	15	58	26
1930	27	99	27
1931	10	45	22
1932	29	104	28
1933	21	64	33
1934	31	85	36
1935	42	99	42
Totals	327	1195	Average 25

During the twenty-one-year period 1915-1936 there has been a well-sustained increase in the ratio of the use of iodine among cases of attempted suicide admitted to the Boston City Hospital (Table 1).

Three hundred and twenty-seven of 1195 attempts were due to iodine ingestion. This represents an average of 25 per cent, and about 16 cases

annually. From 3 admissions in 1916 (8 per cent of suicidal cases) the number has increased annually, with few exceptions, to 42 admissions in 1935.* The number for 1935 represents 41 per cent of all suicidal attempts. When a decrease in the number of iodine cases occurred there was a correlative decrease in the total number of suicidal admissions. The years 1920, 1928 and 1929 were exceptions. The use of iodine for suicidal purposes is probably more widespread among the general population than these figures indicate, but in view of the medical examiner's statement as to the absence of fatal cases and to the fact that the attempt is often concealed by the patient, friends or relatives, we cannot tell how frequently such attempts occur. Doubtless many cases are treated elsewhere or never require hospital care. The continued increase in iodine admissions shows a trend quite similar to the increase noted for alcoholic patients[11] during the same years, and common factors may condition this change.

SEX AND AGE DISTRIBUTION

In this group of patients there were twice as many women as men (Fig. 1). This is noteworthy, for in the total series of suicides studied, female cases slightly exceeded male cases in the ratio of only 1.16:1. In Boston, the ratio of males to females in the total population is approximately equal. The United States Bureau of Census Reports[12] indicate that men are more often successful in suicidal attempts than are women. As was pointed out in a previous study,[1] men prefer more violent and, so, more effective means, while women are given to the more dramatic and less effective ones. Women, spending more time at home than do men, find iodine close at hand in medicine closets and resort to its use as a readily available means. The sex distribution is quite different from that seen in a group of alcoholics treated in the Boston City Hospital between 1915 and 1936, in which men predominate.

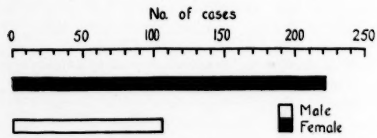

Figure 1. *Chart showing the sex distribution in 327 cases of attempted suicide by iodine ingestion.*

Iodine is especially preferred by young persons as a means of suicide. Figure 2 shows that women attempted suicide by iodine ingestion at an earlier age than did men. The peak years among women are between fourteen and twenty, the numbers decreasing subsequently through twenty-one to twenty-five and then more markedly to thirty-five, after which they become almost negligible. Among men the peak years are between twenty-six and thirty, and after that the number is very small. Ellis[13] states that suicides among women decline after thirty when the period of most intense emotion has passed, whereas in men the incidence increases progressively through life. This statement is substantiated only for women in our series. Thus it is seen that iodine is used chiefly by essentially immature persons at ages when they have failed to gain attention and satisfaction and bid for these by sensational means. Life goes on for most of them about as it was before the attempt at suicide, and they soon may be enmeshed once more in the vicious pattern from which they sought escape. The figures for age distribution among the group of alcoholics requiring hospital care in the same period[11] show that the bulk of cases of alcoholism are seen in the years between thirty and fifty (although it must be remembered that in this group there are countless readmissions). Doubtless the mechanism which motivates both groups is similar, namely, instabilities of character and personality in conflict with and in maladjustment to various psychological and social factors, and inability to resolve situations created often by the conscious act of the subject. The chief difference lies in the means chosen for dealing with conflict in each age group.

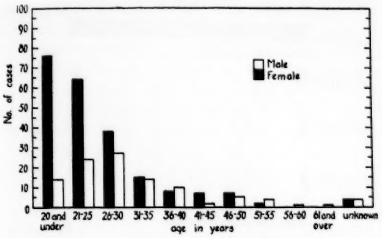

Figure 2. *Chart showing the age-sex distribution in 327 cases of attempted suicide by iodine ingestion.*

Marital Status. As was seen among alcoholics, the largest number of patients in the group attempting suicide with iodine were either single or married and few were divorced, separated or widowed (Fig. 3). Reference to the chart of age and sex distribution (Fig. 2) suggests that the large number of persons who attempted suicide in comparatively early adulthood, that is, before the age

*The number of annual hospital admissions during the same period had approximately doubled (17,174 in 1916 and 37,761 in 1935).

at which marriages are usually contracted, may explain the low incidence of attempted suicides among parties to a broken marriage. Since persons who do not adjust to life before marriage

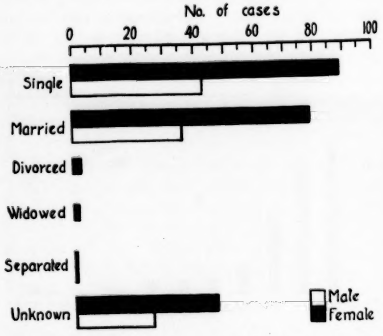

Figure 3. *Chart showing marital status in 327 cases of attempted suicide by iodine ingestion.*

seldom adjust thereafter, the marital state may be discounted as a significant factor, although marital disturbance is often advanced as a motive for suicide. Forty-three men and 89 women were unmarried and essentially the same numbers were married, there being 36 men and 79 women in the latter group. Two women were widowed and 3 were divorced, but no men were represented in these groups. The implication from this may be that a broken marriage is less traumatic to men than to women, who feel insecure and forsaken when the support of a partner is withdrawn, however inefficient he may have been. The marital status of 26 men and 47 women was not ascertained. Undischarged energy is low in widowed persons, who may seek and sometimes find substitutes for lost mates, although it has been noted[14] that among first admissions to mental hospitals in Massachusetts the incidence of divorced persons is high.

Color. White persons predominated in the group, there being 81 white men and 163 white women. Four male and 19 female Negroes are included. The color of 21 men and 39 women was not ascertainable from the records.

Occupational Status. It was in many cases impossible to learn the occupation of these patients; 27 men and 47 women failed to state their occupation at the time of the attempt. Few persons in the group were employed in highly lucrative positions. Fifty-three of the women were housewives and 42 were domestics, while 33 were unemployed. Eleven were skilled workers or professional persons and 8 were students. Twenty-eight were employed in trades requiring no particular skill. The situation was similar among the males represented. Of these, 32 were skilled workers or were employed in professional capacities, but the majority were in the lower occupational brackets. Six were unemployed and 1 was a student. Seven were in domestic service and 17 were unskilled. There were 16 laborers in the group. The majority of these persons were in marginal or dependent circumstances. The discouragingly small return from their labors may have been a factor in the production of their suicidal attempts. The order of size of these groups is not significantly different from that seen in the total suicide group studied, and probably bears no relation to the use of iodine for suicidal purposes. Rather it is a commentary upon a social order which fails to supply adequate opportunity for work and a reasonable financial return to some of its members. Economic stress and its sequela, and domestic strife, rank high among stated motives for suicide.

Motives. Suicides are very loath to admit the motive for their act, and unless the inquiry is tactfully made it usually yields no information. Domestic trouble was the most important single factor in producing a suicidal attempt (Table 2).

Table 2. *Motives for Attempted Suicide by Iodine Ingestion.*

MOTIVES	MEN	WOMEN	TOTAL
Domestic troubles	12	44	56
Unemployment	6	5	11
Ill-health	—	9	9
Economic reasons	4	4	8
Love affairs	2	6	8
Miscellaneous	5	4	9
Unknown	77	149	226
Totals	106	221	327

Unemployment of self or marital partner, ill-health, economic reasons and turbulent love affairs ranked next in order. Abuse by a drunken father, and family and school difficulties were stated by a number of individuals to be the cause of their attempt. One woman used iodine for the effect it would have in bringing an errant lover to heel. Among the specific causes grouped under ill-health were several cases of pregnancy among unmarried females; the rest were physical and mental diseases of chronic nature. One man admitted his attempt was for a sensational purpose solely. A considerable number of persons (6 per cent) were alcoholic at the time of their attempt, but this is considered to be a predisposing circumstance rather than a motive. By far the largest group gave no reason for their attempt and the inquiry was not pressed by the attending physician. One woman stated that she made her suicidal at-

tempt after learning that she had a venereal disease. One man was a drug addict. The known motives in this group are about the same in the group of attempted suicide by various other means.

Treatment. Treatment of these patients was for the most part routine by the medical services, and usually consisted of gastric lavage and the oral administration of starch solutions as an antidote. Two persons were treated for iodine poisoning on the surgical services, but they were suffering from the effects of slashing as well as from iodine ingestion, and their most pressing need was treatment of their wounds. Stimulants were given to some patients suffering from collapse. Thirty-seven patients were treated in the out-patient department and were not seen until the acute stages had subsided. Eight persons were treated only with rest and observation.

Condition on Discharge. As might be expected the majority of cases were discharged "relieved": 57 men and 141 women were so adjudged on leaving the hospital. One woman was "not relieved"on discharge. Twenty-two men and 35 women were discharged on their own request against advice.* No data are ascertainable as to the condition of 20 men and 41 women on discharge. Six men and 3 women required further care and received it at other institutions, chiefly mental hospitals. One patient was dead on discharge, but he was a sixty-year-old man who had taken iodine and bichloride of mercury at the same time. Death in this case was due to renal failure associated with the bichloride poisoning.

DISCUSSION

The use of iodine in suicidal attempts does not of itself indicate any psychologic characteristic that sets the user apart from those who attempt suicide by other means. The foregoing data indicate that, of all poisons used for the purpose of suicide, iodine is most commonly employed, but it is less efficient than most other methods in accomplishing the desired aim. It is noteworthy that undue importance is attached to iodine by the general public; this is possibly due to the prominent poison label which is always attached to the container when dispensed by a drugstore.

There is a certain cathartic benefit in non-fatal suicidal attempts, and even if life is not destroyed, the rehearsal of the emotions and the behavior entailed may often serve to weaken the wish to die. There are no available data to show how many of the patients included in this study were readmitted later to this or another hospital because of another suicidal attempt. However, it is clear that the suicidal individual is, by virtue of his act, in need of therapeutic assistance in order to cope with the difficulties which prompted his deed.

Stearns[15] has pointed out that a suicidal attempt may be a symptom of an advanced mental disease. Thus, it follows that these patients have in the past often failed to receive treatment for the most pressing of their needs. The condition of some of them is comparable to that of a medical patient who might be superficially treated for furunculosis when one had failed to ascertain and treat the basic and underlying diabetic condition which had produced the boils. Likewise, it is not unreasonable to suppose that these non-fatal suicide patients have required the services of this or another hospital or social agency at some later period because they were not treated for the personality disturbance while they were first hospitalized.

*These persons usually left immediately after admission and refused treatment.

CONCLUSIONS

During a twenty-one-year period (1915-1936) 27 per cent, or more than one-fourth of all the patients admitted to the Boston City Hospital after suicidal attempts had ingested iodine. The number of these cases was 327 out of 1195 attempted suicides. There has been a definite increase in the use of iodine in suicidal attempts greater than the increase in suicidal admissions.

Not one death due to iodine poisoning occurred among these patients.

The use of iodine in suicidal attempts is preferred chiefly by young women. There were approximately twice as many women as men in the group. Among the women the great majority of patients were under twenty-five years of age, and among men the majority were under thirty-five years of age.

Single and married persons in the group were about equally distributed.

Although these persons were disturbed individuals, and many of them were suffering from definite mental conflict, nothing other than symptomatic treatment was attempted in the hospital.

As to occupational status, these patients were chiefly in the lower economic brackets. Few patients came from groups above a marginal or dependent economic status.

Domestic discord, unemployment, ill-health, economic troubles and unhappy love affairs were commonly stated as motives. Most stated motives involved a lack of social and psychological adjustment.

On discharge, most patients were relieved of their physical symptoms. No information is available as to their mental condition or subsequent course after discharge.

I am greatly indebted to Miss Ruth Church, Doctor Mildred Gray, Miss Ethel Pokross and Miss Sumiko Oura for help in preparing this material.

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MENIERE'S SYNDROME: DIFFERENTIAL DIAGNOSIS AND TREATMENT

Case Report

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WINCHESTER, MASSACHUSETTS

IT IS the purpose of this paper to present a case in which the differential diagnosis is of unusual interest and also to illustrate a case of Ménière's disease in which the surgical treatment was highly successful.

The syndrome of Ménière's disease was first described by a Frenchman of that name in 1861 as a series of attacks of terrific dizziness, nausea, vomiting and tinnitus, associated with unilateral deafness of a progressive nature. The unilateral deafness and tinnitus usually differentiate the dizzy spells of Ménière's disease from those of arteriosclerosis and other causes. Attacks of so-called pseudo-Ménière's disease may be exactly similar to those of Ménière's but lack the unilateral deafness; it has been thought that these may represent an early form of true Ménière's. In the first description of the condition, before the days of bacteriology, the pathological picture was confused with acute labyrinthitis, and a purulent hemorrhagic exudate in the labyrinth was reported. Textbooks still speak of Ménière's disease as due to hemorrhage into the internal ear, but today the condition is thought of more as an explosion of the function of the inner ear, possibly due to a lesion of the auditory nerve. Actually the etiology and pathology are unknown. No necropsy of a true case of Ménière's disease has been reported.

CASE REPORT

The patient was a lawyer, aged 56, at the time when first seen on October 22, 1933, at his home. The previous day he had played a very strenuous game of golf, after which he was somewhat fatigued and had a slight headache. He slept well during the night, arose about 9 a. m., and while taking a shower before breakfast, suddenly felt extremely weak, dizzy, faint and nauseated. He went to bed immediately and was seen within a very short while. The patient had no pain of any sort. His bowels had last moved the day before. The past history revealed that in the previous 2 years the patient had had three or four attacks of "acute indigestion"; that is, attacks of pain developing in the mid-epigastrium, localized and lasting for 1 or 2 hours, relief being obtained promptly by an enema. He was always perfectly well on the following day. His past history was otherwise noncontributory.

Physical examination showed a well-developed and well-nourished, thick-set type of man. He was obviously in severe shock with a drenching perspiration. His color was an ashen gray. He was very cold and clammy. He showed no nystagmus, eye motions were normal, and the fundi were not remarkable. Ears, nose and throat appeared normal. The lungs were clear and resonant. The heart did not appear enlarged, was regular in action at a rate of 80, and sounds were of fair quality. The blood pressure was 130/80. The abdomen was soft and showed no tenderness. The extremities were not remarkable, reflexes being equal, active and normal. There was no edema. The respirations had become distinctly depressed, shallow, slow and slightly irregular. The patient appeared to be in a critical condition.

Caffeine sodium benzoate was given, followed by some improvement in respiration, although the patient had sunk into a semi-stuporous state and the blood pressure had shortly fallen to 110/80. It was thought at this time either that the patient was in a profound state of shock from a cerebral vascular accident of which no reflex manifestations could be found, or particularly in view of the previous attacks of "acute indigestion," that he was suffering a coronary thrombosis without pain. Within another half-hour the cardiac rhythm had shifted to a complete irregularity, apparently fibrillation. This seemed further confirmatory evidence of a silent coronary thrombosis. Fifty cubic centimeters of 50 per cent glucose was administered intravenously.

Shortly thereafter the patient was seen in consultation with a Boston internist, who agreed with the diagnosis of coronary thrombosis. Two thousand cubic centimeters of

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normal saline was given by hypodermoclysis. Morphine 1/6 gr. was given for restlessness. During the afternoon the patient began to look slightly brighter and stopped perspiring. Later that afternoon the auricular fibrillation ceased. The following morning the patient appeared greatly improved, and showed good heart sounds at a rate of 76 and a blood pressure of 135/90. His temperature was 99.5°F., and he showed a white-cell count of 16,000. His urine showed no albumin or sugar and a normal sediment. He complained of some slight discomfort during this day in the region of the left shoulder blade. There was no evidence of congestive failure.

Assuming the diagnosis to be coronary thrombosis, the patient was kept in bed for the following 5 weeks, and resumed part-time work after an additional 3 weeks. He apparently made a completely uneventful convalescence.

On January 26, 1934, 3 months after the first attack, the patient had a slight feeling of epigastric distress, suddenly became quite weak, felt very dizzy and faint, and promptly went home. He had had a slight head cold the previous day and had had no bowel movement for over 24 hours. He was seen by the writer shortly after his arrival home. At this time his skin was pale and cold but showed no profuse sweating. The heart sounds were regular; the blood pressure was 118/70. Following the application of heat to his body, an enema and a few hours of complete bed rest, the patient felt quite normal. After 2 days of rest he insisted on returning to work.

On April 10 the patient again had a similar episode after his noonday meal, with general weakness, dizziness, nausea and profuse vomiting. He came home and went to bed, but the dizziness, nausea and vomiting continued. In the interval between this and the previous attack he had felt quite well. Examination on this occasion again showed a moderately shocked man, but without the profuse sweating of the first attack. His heart was again grossly irregular in rhythm, with an apical rate of 110 and a radial rate of 70. The heart sounds were of fair quality. The blood pressure was 115/80. One-fourth grain of morphine was given hypodermically and symptoms ceased about a half hour thereafter. The following morning the patient felt quite normal and the heart showed a normal rhythm at the rate of 60.

At this point the question arose whether these recurrent spells could represent anginal equivalents or small coronary thromboses, or if they might possibly be a manifestation of primary paroxsymal auricular fibrillation. It seemed that the patient was too weak at the moment and too well directly after the attacks for these minor seizures to actually represent coronary occlusions.

On April 18 the patient was seen in consultation by a Boston cardiologist. The following is an extract of his report:

> I was not sure about the diagnosis, except for paroxysmal auricular fibrillation. This irritation of his heart seems to me to have been more of a reflex to some other trouble than primarily the cause of his attacks, although the latter is possible. The diagnosis rests between coronary disease with an attack of coronary thrombosis last October, gallstones and cerebellar vascular disorder. I am a little more in favor of one of the last two diagnoses than of the first-named, although it is quite possible that he may have had two different conditions, such as I have occasionally encountered in puzzling cases; that is, he may have gallstones or a cerebellar vascular condition complicating the more pronounced attack, namely the coronary thrombosis in October.
>
> His headache, dizziness and nausea have been out of proportion to the pain, and in that respect different from the usual attacks of coronary origin.

An electrocardiogram taken at this time showed a normal rhythm at the rate of 80, with flat T waves in Lead 1, low T waves in Leads 2 and 3 and rather low voltage of QRS waves with slight left-axis deviation. Fluoroscopic examination showed a full-sized, horizontally placed heart. By orthodiagram the transverse diameter of the heart measured 12.2 cm. and the internal diameter of the chest was 25.5 cm.

Following this a Graham test was done and showed normal appearance and function of the gall bladder. A gastrointestinal x-ray series and a flat plate of the abdomen were normal.

On April 27 the patient felt well on awakening. While walking to the railroad station he began to feel slightly dizzy, and grew more so while on the train going to Boston. He walked about a half mile to his office in town, growing progressively dizzier and weaker. On reaching his office he became extremely nauseated and vomited. The patient was seen at his office in Boston. His pulse was 72 and regular; the blood pressure was 140/100; the eyes for the first time revealed a nystagmus on looking to the left and upwards. The patient was taken directly to a Boston hospital and put to bed. On arrival there an electrocardiogram was immediately taken, which showed a tracing similar to the previous one except that the T waves in Lead 1 were definitely upright. After arrival at the hospital the blood pressure dropped to 100/60 and the patient appeared in severe shock. The nystagmus persisted during the afternoon. Morphine, ¼ gr. given hypodermically, brought relief of the unpleasant symptoms. The following day the patient felt as well as ever and showed nothing remarkable on physical examination, including a careful neurological examination. A blood Hinton test was negative.

At this point, having seen the patient on several occasions without any cardiac irregularity, we became more fully convinced that the situation was not primarily cardiac and wondered if Ménière's disease might be the answer to the problem. The patient was seen by an aurist, who agreed that Ménière's disease was a distinct possibility. The patient remained at the hospital for 4 days. Labyrinthine function tests showed marked hypofunction in both ears, but more marked on the left. Audiogram showed nerve type of deafness in both ears, somewhat greater on the left. Sinus plates for possible foci were negative. Dental films showed four apical abscesses, and two of the involved teeth were removed in the hospital; he returned home within a few days, and the other two were taken out the next week at the dental surgeon's office. The basal metabolic rate was —13; because of this it was decided to put the patient on thyroid extract (Armour), 1 gr. daily.

On May 16 the patient awoke with a complete loss of memory for events of the previous 2 to 3 months. The week before, the other two abscessed teeth had been removed, and the socket of one was still draining. General examination was essentially as before, with blood pressure 110/90 and a regular pulse of 80. The patient was returned to the hospital. Later that afternoon his mind entirely cleared, and he again seemed to be in a normal state. Dental x-rays were repeated and showed one more infected apex, and the tooth was removed. Skull plates which were taken proved entirely normal. After 2 days in the hospital the patient was discharged, apparently comfortable, on no medication. The spell of

amnesia was quite unexplained, other than a possible toxic manifestation from the dental sepsis or a possible peculiar reaction to thyroid.

On September 18 the patient had his next episode of vertigo, nausea, vomiting and weakness. During the summer he had been quite well up to the previous day, when he had developed an acute gastrointestinal upset which had also affected other members of his family. The patient re-entered the hospital. Examination at this time was as before with a normal heart rhythm and with nystagmus more marked toward the left. Again, after ¼ gr. of morphine, symptoms subsided in a short while.

On November 22 the patient arrived at his home with a recurrence of a spell exactly like the previous one. Following this attack the patient was seen by an ophthalmologist who reported a completely normal ocular examination, including visual fields.

On February 1, 1935, the patient suffered another attack exactly like the previous one. At this time he reported that he had felt somewhat dizzy off and on during the previous 2 weeks, and as though he were going to fall toward the left side. He also noted that there was more tendency toward dizziness with sudden movement of the head or while in motion as on the train.

In May the patient was having some increasing dizziness, especially in the morning on arising. He also complained of dull pain in the occipital region of his head. The patient was readmitted to the hospital, where he was seen in consultation with a neurologist, who reported a completely normal neurological examination with the exception of nerve deafness in both ears, greater on the left. A lumbar puncture showed clear, colorless fluid with an initial pressure of 170 mm. of water and normal dynamics. After withdrawal of 10 cc. the pressure was 105 mm. Examination of the fluid showed no cells; a total protein of 33 mg. per cent; a negative test for globulin, a goldsol curve of 0011100000 and a negative Wassermann.

On June 1 the patient had another attack of vertigo similar to the previous ones. While he kept his eyes closed the dizziness was much less. There was marked nystagmus to the left. Following this attack a trial of the Furstenberg[1] low-salt diet with ammonium chloride was started.

On July 1 and again on July 22 there were recurrent major attacks of vertigo with mild vertigo between them. Apparently the low-salt diet which the patient had followed carefully had been of no benefit.

The situation was then discussed with the various consultants who had seen the patient; it was the consensus that we should make a diagnosis of Ménière's disease and that the various factors tended to incriminate chiefly the left eighth nerve. Having been favorably impressed by Dandy's recent reports[2, 3] we decided to communicate with him regarding the possibility of hemisection of the left eighth nerve. He agreed with its probable feasibility. The patient was at the point of distraction with the constant discomfort and interruptions to his professional work and agreed to submit to the operation.

On September 1 he went to Baltimore, where on September 6 a hemi-section of the left eighth nerve was performed under Avertin and ether anesthesia. The patient made a normal operative convalescence and returned to Boston on September 21.

Up to the present time, three years after operation, the patient has had no recurrence of his incapacitating spells of vertigo. There has been slight dizziness at times but never any of such degree as to incapacitate the patient for his work, and no associated nausea or vomiting. Since operation there has been slight increase in deafness on the operated side. There has been no evidence on history or examination of further cardiac disturbance.

DISCUSSION

The complete lack of pain in the patient's first attack, while by no means ruling out a coronary occlusion, is a point against this diagnosis. The appearance of pain behind the left shoulder blade the following day is a point in favor of it. Likewise the flat T wave of the electrocardiogram in Lead 1 taken six months later points toward coronary disease. The rise in the white-cell count following the first attack is consistent with a thrombosis. The complete absence of dyspnea and other signs of myocardial failure in an attack with such severe shock seems unusual in coronary disease. Furthermore the patient has since led an active life, without any suggestion of heart weakness.

The occurrence of paroxysmal fibrillation in the first and third attacks is of interest. Orgain, Wolff and White[4] have pointed out that reflex paroxysmal fibrillation may occur with various visceral or emotional disturbances without any primary heart disease. In the present case the fibrillation might well have been reflex to an acute labyrinthine disturbance. It is not possible to state dogmatically whether the patient did or did not have a coronary thrombosis when first seen. In the light of subsequent developments, it seems most likely that the earlier as well as the later attacks represented Ménière's disease.

It is of interest that tinnitus was entirely lacking in this case. In 42 cases reported by Dandy[3] tinnitus occurred in all but 1, and in the past was considered an essential to the diagnosis. In this case a conscientious trial of the Furstenberg dietary regime for a period of two months was without benefit. Dandy states: "The eradication of infected foci has not altered and cannot alter the course of the disease." This statement is borne out in the above case. The patient also demonstrates the extreme degree of shock and prostration which may occur with attacks of Ménière's disease.

From October, 1933, through July, 1935, this patient had ten major attacks, coming with somewhat increasing frequency, and making life quite unbearable. Since the nerve section in September, 1935, there have been no further major attacks. From the patient's point of view and from ours the operative results have been highly gratifying.

Dandy's report[3] of 42 cases states that no known attacks have occurred subsequent to operation. He further states: "The relative frequency of Ménière's disease will doubtless be determined with greater accuracy two or three decades hence.

At present the diagnosis is all too frequently missed, and in addition lack of knowledge concerning the value of operative treatment has prevented the same concentration of cases that obtains in other well-recognized conditions with a definite form of treatment." In this report there were 10 cases in which various, previous operative procedures had been performed — based on wrong diagnoses: (1) cholecystectomy and appendectomy, (2) cholecystectomy, (3) tonsillectomy, (4) exploration for cerebellar tumor, (5) tonsillectomy, (6) cholecystectomy and appendectomy, also tonsillectomy, (7) tonsillectomy and appendectomy, (8) tonsillectomy and cholecystectomy, (9) mastoidectomy and all teeth removed, (10) fallopian tubes and ovaries removed.

In the case here reported the diagnosis of Ménière's disease was reached only after a year of thorough study in co-operation with a number of consultants. This extremely distressing condition may masquerade in deceptive guise and be accompanied by associated signs and symptoms which should not obscure the true underlying condition. Among these have been reported bradycardia, fainting spells, feeling of pressure in the lower chest, dark spots in the visual field, diplopia, headaches of varying types, associated migraine, tightness in the back of the neck, pains in the ears, intense desire to defecate and urinate and unconsciousness. To these we may add paroxysmal auricular fibrillation and an interpolated spell of amnesia. The association of these phenomena of the sympathetic and central nervous systems are curiously akin to the collateral symptoms seen in migraine patients, and lend some weight to the suggestion that these may be fundamentally related conditions.

Dandy[5] has now operated on 223 cases without a death from any cause. Munro,[6] in an excellent and comprehensive review of the subject, collected reports of 75 well-authenticated cases in the literature (including 42 of Dandy's) operated on in various sections of the world, all showing definite cures. He reported 5 cases of true Ménière's disease in which a vestibular-nerve section was done with 100 per cent relief. He also reported another patient on whom operation was planned following failure of a Furstenberg diet, and it is interesting to note that this individual showed a classical syndrome but lacked the tinnitus, as did our patient. Horrax[7] reported briefly 32 patients with "fairly typical Ménière's syndrome. . . . Twenty-four of these have on the whole responded well to the Furstenberg diet for periods they have so far been followed — one or two months up to two years. A few could not be followed or were not helped." Eight patients were operated upon by nerve section with no mortality. All but 1 of these were completely cured. "One patient had little or no relief. In this instance the diagnosis may have been wrong or the condition of too long standing to be affected favorably."

Since a relatively safe operation may bring complete relief to these sufferers, it behooves us all to be more on the outlook for this condition than we have in the past.

SUMMARY AND CONCLUSIONS

A case of Ménière's disease without tinnitus is reported in which the diagnosis was at first confused with coronary thrombosis.

A trial of Furstenberg's low-salt diet was without avail.

Following ten major seizures in nineteen months, complete relief has now continued for three years following a hemi-section of the left eighth nerve.

The diagnosis of Ménière's disease may be confusing and masked by secondary symptoms.

In a large series of cases hemi-section of the eighth nerve has proved a safe and effective method of relieving sufferers from Ménière's disease.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 24371

PRESENTATION OF CASE

A sixty-five-year-old, native, white male entered the hospital complaining of shortness of breath and pain between the shoulders.

One year before entry he began to have wheezing respirations, more pronounced at night, associated with a nonproductive cough. Nine months before entry he developed increasing dyspnea which interfered with his work, and the cough became productive of small amounts of mucoid sputum. He also noticed that the range of his singing voice was somewhat lessened. His physician prescribed cough medicine and some pills to be taken three times a day for irregular heart action. His cough and wheeze improved somewhat but three months before entry he was transferred from manual labor to clerical work because of increasing dyspnea and weakness. A physical examination by his physician two months before entry revealed a few rales at the bases of his lungs and a regular heart rhythm. Medication was omitted at that time. One month before entry a sharp, unremitting pain between the shoulder blades suddenly made its initial appearance. The pain was aggravated by deep inspiration, but did not interfere with sleep. At times it was associated with a pleuritic pain in the left lower anterior chest. At that time he also began to raise small amounts of "old blood," usually in the morning. Occasionally late in the afternoon he raised a little bright red blood. Ten days before entry a physical examination by his physician revealed an essentially normal heart and lungs. Five days before entry the pain in his chest became so severe that for the first time he did not go to work and was bad enough to keep him from sleeping except with soporifics.

His family history and past history were noncontributory.

Physical examination revealed a barrel-shaped chest, resonant throughout, with audible wheezes in both lungs. Posteriorly over the fourth, fifth and sixth ribs on the left, questionable bronchovesicular breathing and increased breath sounds could be heard. The left diaphragm was slightly elevated. The ribs over this area were somewhat tender. The heart sounds were distant but regular, and the blood pressure was 130 systolic, 80 diastolic.

The temperature was 100°F., the pulse 95. The respirations were 20.

The urine examination was negative. The blood showed 4,050,000 red cells with 70 per cent hemoglobin, and 8500 white cells with 62 per cent polymorphonuclears. The nonprotein nitrogen of the blood serum was 28 mg. per cent and a Hinton was negative.

On the seventh and eighth hospital days 700 cc. of air were put into his left pleural cavity creating a pneumothorax. X-rays on the eighth day showed a round, slightly lobulated mass, about 9 cm. in diameter, in the left upper lobe, slightly posterior. A few questionable small cavities were seen in the mass. There was a pneumothorax present on the left side which separated the lower lobe from the parietal pleura laterally and along the mediastinum. Only a minute amount of air was visible about the apex. There was only slight decrease in the aeration of the left lung. This was because of the small pneumothorax present. On the tenth day an operation was performed.

X-RAY INTERPRETATION

DR. AUBREY O. HAMPTON: We have two films, one taken before and one after the pneumothorax was induced. They show a round, rather ovoid mass apparently within the left upper lobe. It is smooth except for a few lobulations on the anterior aspect and laterally.

DR. JOHN W. CASS: The report says that a few questionable small cavities were seen in the mass.

DR. HAMPTON: There are two areas of diminished density about 1½ cm. in diameter which may be confused with the air in the pleural cavity, but they do not appear to be in the pleural cavity. I would say that there is possibly a cavity in the mass. The left lung root is partially obscured by the mass. I see no enlarged glands and no calcium. He has a barrel-shaped chest and a low diaphragm, with a few emphysematous blebs in the right apex.

DIFFERENTIAL DIAGNOSIS

DR. CASS: The early part of the history suggests a cardiac situation but the cardiac element slowly leaves the picture as you go on. Certainly wheezing and unproductive cough coming on at night are manifestations of a failing left ventricle. However, three months after the onset, although the dyspnea had increased, he began to raise mucoid sputum which would not be compatible with increasing heart failure. With the latter, we would expect it to be frothy and blood-tinged, whereas mucoid sputum suggests that the primary pathol-

ogy is within the lungs. Why the range of his voice changed, I am not sure. It could not be connected with any primary heart situation. It might possibly be related to mediastinal disease or trouble with the upper portion of either lung. The irregular heart is probably of no significance in this picture. The most common irregularity at this age would be the arrhythmia of extrasystoles, or fibrillation. Six months after the dyspnea increased markedly he became very weak and had to change his work from manual labor to clerical work, but in spite of temporary improvement there was no definite change in the situation. Eight months before entry for the first time he began to have pain located between the shoulder blades. It involved the pleura on the left side but did not radiate. It is hard to tie this up with the heart situation unless he had an aneurysm or pericarditis. So we are getting more and more certain that we are dealing with pulmonary disease.

The next thing was the raising of blood. It seems to have been old blood, which is interesting. Evidently he did not have real hemorrhage. He must have had oozing, and if it was from the lung we could imagine that it was oozing into a cavity and was raised only when he awoke in the morning. Such blood, of course, could come from the mouth. It probably could not come from the intestinal tract. We have no description of the digestive tract. The bright blood in the afternoon shortly before he came to the hospital would suggest that the lesion was by then actively bleeding. Ten days before entry physical examination of the heart and lungs was reported as normal. We would suspect that they should have found something at that time. The symptoms became very severe five days before entry, and he had to stop work. The disease was very rapidly progressive, especially with regard to the pain during the last five days before he entered the hospital. I doubt very much that pain associated with any heart condition or any mediastinal syndrome of this degree would allow him to work for nearly a month. At the end of the history we are certain that the primary disease is not in the heart but most likely in the lungs, and that with this history in a man of sixty-five a malignant tumor in the lung would be the most likely diagnosis. It would be possible, of course, for a chronic abscess to give these symptoms, but we have no history of any etiologic factor and no history regarding any great amount of sputum.

The physical examination was negative except for evidence of emphysema and physical signs which are compatible with a mass in the left upper chest, or with consolidation, or even with bronchial obstruction and secondary collapse of the lung. The fact that the diaphragm was high is additional evidence that there was some element of collapse, and the fact that the ribs were tender is interesting. They are often tender in conditions involving the pleura. The heart was within normal limits. So the physical examination localizes the lesion, — a mass in the upper lobe of the left lung, — but does not necessarily rule out an abscess.

So far as the laboratory data are concerned, he had a little fever, but not too much to fit a noninfectious process, and the blood picture does not suggest infection. It is surprising there was so little anemia. His kidneys were in good shape, judging from the normal nonprotein nitrogen and the one normal urine examination.

The diagnosis depends then primarily on the x-ray. Pneumothorax in such situations is of great diagnostic value in that it outlines masses. If a mass is present, it helps to locate it, and also determines the condition of the surrounding structures and the point at which it can be most easily approached. Of course one should be cautious in its use if abscess were suspected. We would be concerned about rupturing the lesion.

The x-ray with the aid of pneumothorax definitely localized a mass in the upper lobe of the left lung, and it pretty well ruled out the possibility of abscess. There are some questionable cavities but we are not sure of these. I should think the first impression would be a malignant tumor. Malignant tumors may hollow out in some degree, resulting more often in multiple small cavities, but occasionally developing a single large cavity so that it is impossible by x-ray to differentiate abscess and tumor. Sarcomas are more liable to do this than carcinoma, but carcinomas do occasionally form cavities. Though it is possible that this is a benign tumor, the bleeding would tend to put it into the group of malignant tumors. I should think that a malignant tumor was most likely. We have no evidence that adjacent glands are involved and no evidence that we are dealing with a metastatic tumor. We have to assume that it is a primary condition. We should mention the possibility of cyst — nothing so far definitely rules it out. A primary dermoid in a man of this age is very rare, and on the law of chances I should rule it out. Echinococcus cyst is very rare and there is no evidence here to permit us to make that diagnosis. Very often when cyst is suspected the sputum is of great help. We have no sputum studies here to aid us. So as far as we can go is to say that this is probably a malignant tumor and, because of the age, the bleeding and the sequence of events, it is most probably an adenocarcinoma of the upper lobe of the left lung.

Dr. Donald S. King: I think this is a good case to illustrate the symptoms of so-called pulmonary carcinoma, to bring out the fact that it is actually bronchiogenic, because this patient started with

wheeze, which is so characteristic, later developed dyspnea and severe uncontrolled pain and hemorrhage. It is impressive as we go over our 158 proved cases now to see how common this sequence of events is.

Dr. Edward D. Churchill: We made a diagnosis of primary carcinoma of the lung, and at operation I removed the entire left lung. We realized he was a poor operative risk. He did surprisingly well so far as respiratory symptoms were concerned. He developed an interesting cardiac arrhythmia, however, that I think Dr. Comeau will speak about.

Dr. Wilfrid Comeau: This man developed a queer ectopic tachycardia in the nature of paroxysmal tachycardia or impure flutter with a-v block. We digitalized him with large doses and at times his rhythm would return to normal.

There was never any evidence of heart failure during the time I followed him, so although the rapid rate may have contributed to his death I doubt very much that heart failure was the fundamental cause of death. The electrocardiogram did suggest some pericarditis which may have been part of the reaction to the operation. At the time it was largely a matter of controlling the cardiac rate. His heart was never embarrassed functionally.

Clinical Diagnoses

Carcinoma of lung.
Emphysema.
Pericarditis.
Bronchopneumonia.

Dr. Cass's Diagnosis

Bronchiogenic carcinoma, left upper lobe.

Anatomical Diagnoses

Epidermoid carcinoma, grade IV of bronchus of left upper lobe with metastases to liver, brain and kidney.
Pulmonary emphysema.
Fibrinopurulent pericarditis.
Operative wound: left total pneumonectomy.
Pleuritis, acute operative, left.
Acute duodenal ulcers.
Benign hypertrophy of prostate.

Pathological Discussion

Dr. Tracy B. Mallory: The lung which Dr. Churchill resected showed a single large nodule of tumor 9 cm. in diameter occupying the major part of the upper lobe. It extended nearly to the floor of both the mediastinal and the lateral aspect and stopped short at the interlobar fissure on its inferior margin. On dissection it was found that a bronchus 4 mm. in diameter, one of the second divisions of the main bronchus to the upper lobe was completely occluded by the tumor and this we may reasonably conclude was the primary site. On microscopic examination the tumor proved to be a squamous-cell carcinoma of a very low degree of differentiation and therefore of a high grade of malignancy. It was interesting that none of the bronchial glands, either those resected with the tumor or the remaining ones which were examined at autopsy, showed any evidence of metastasis. Nevertheless, the tumor had metastasized very widely — far more so than anything in the clinical course of the patient would have led one to suppose. The liver contained a great many metastatic nodules and small metastatic lesions were found in the brain and in one kidney.

The immediate mechanism of death was not easy to determine. In the last twenty minutes of the patient's life he developed a very sudden and severe dyspnea which suggested to the intern who saw him during this attack the possibility of a pulmonary embolus. This was not found and there was nothing else which would explain a sudden attack of that character. The most important lesion seemed to be an acute fibrinous pericarditis. The left pleural cavity seemed to be in excellent condition from the surgical point of view. It contained a considerable amount of gelatinous exudate but no pockets of pus or evidence of an important degree of infection. The end of the severed bronchus had been covered with a graft of lung tissue which had become firmly attached and showed evidence of organization. His remaining lung, however, did show a very severe grade of structural emphysema with many blebs varying from 2 to 8 mm. in diameter. I am inclined to agree with Dr. Churchill that the most important reason for this man's failure to rally from the effect of the operation was a pulmonary insufficiency.

CASE 24372

Presentation of Case

First Admission. A two-year-old, white, American boy entered the hospital for study because of swelling of his hands, feet and abdomen of four months' duration.

Eighteen months before entry he gradually developed swelling of the backs of both hands and wrists and of the dorsums of both feet. His abdomen became enlarged, and he had a high fever, with prostration and vomiting. He entered an outside hospital where he remained for four months. He continued to run a fever and was on the dangerous list most of the time. Finally he was given a transfusion which resulted in complete and rapid subsidence of all his symptoms. He went home and very quickly began to gain

weight and strength. The following winter he had whooping cough, which he stood very well. Four months before entry he again developed swelling of the hands and feet with enlargement of the abdomen and persistent fever. This time, however, the joints and abdomen were painful. These symptoms persisted unchanged up to the time of entry. The pain on motion in the wrists and ankles was variable, and he often was unable to use his hands to feed himself. Three weeks before entry he complained of stiffness in the left side of his neck, and a slight diffuse swelling appeared over the right side of the neck and face. The stiffness often seemed to interfere with mastication. During his illness he lost a considerable amount of weight.

From the time of birth the patient had never had a normal bowel movement. He always required laxatives or enemas. He had a good appetite, but his diet consisted mainly of potatoes and bread, with adequate amounts of milk, but little meat or eggs. His past history was otherwise negative. There was no history of arthritis or any other similar diseases in other members of the family.

Physical examination revealed a very anemic, undernourished, alert boy lying quietly in bed in no obvious pain or discomfort. The skin was dry and the hair was sparse and worn off the back of the head. There was generalized enlargement of the superficial lymph nodes. Some of the axillary nodes measured up to 1 cm. in diameter. The heart and lungs were negative. The abdomen was protuberant, and there was a small umbilical hernia. Both wrists were definitely swollen, but no fluid could be made out within the joints. They were not tender. Both ankles seemed to be slightly enlarged on their superior surfaces.

The temperature was 101°F., the pulse 130. The respirations were 35.

The urine examination was negative. The blood showed a red-cell count of 4,540,000 with 50 per cent hemoglobin, and a white-cell count of 16,100 with 81 per cent polymorphonuclears. A blood Wassermann was negative and a 1:1000 tuberculin test was negative. Three blood cultures gave no growth. The blood sugar was 82 mg. and the uric acid was 1.5 mg. per cent. X-rays of the affected joints showed thickening of the soft tissues with a moderate degree of bone atrophy. The joint spaces were not narrowed, and there was no evidence of bone destruction or proliferation. There was an abnormally early appearance of ossification centers in the hands and feet. X-rays of the sinuses and teeth were negative.

He remained in the hospital for almost three months and during the entire time ran a septic type of temperature fluctuating between 99 and 103°F., and rarely returning to normal. Repeated urine examinations were negative. The red-cell count of the blood varied between 4,000,000 and 5,000,000 with 50 to 60 per cent hemoglobin, and the white-cell count between 12,000 and 20,000 with 66 to 81 per cent polymorphonuclears. The stools were bulky and malodorous. He was given three transfusions and was treated with liver extract, Brewer's yeast, potassium iodide and aspirin. At one period he was given high colonic irrigations. None of these measures appeared to have any marked effect on his condition. Plaster casts were applied to some of the affected joints, which relieved the pain by immobilization. On several occasions he had skin rashes which were thought to be due to drugs.

During his course three blood cultures, Malta fever agglutination and gonococcus complement-fixation tests were negative. An analysis of the gastric contents showed no free acid and 40.3 units of total acid.

Two weeks before discharge he had a brief attack of apparent bronchopneumonia which cleared promptly. An x-ray of the chest at that time showed the heart to be enlarged and of an abnormal shape. The enlargement was most marked in the transverse diameter and in the region of the right auricle. There was general thickening of the lung markings in both lung fields and the interlobar septum was visible on the right. At the time of discharge the liver was palpable 4 cm. below the costal margin, but the spleen was not felt. There was still generalized enlargement of the lymph nodes.

Final Admission (six years later). After discharge he was admitted to a hospital for crippled children. At the time of admission there, both wrists, elbows, feet, ankles, and knees and the spine were involved in the arthritic process. Various forms of therapy were instituted including medication with cod liver oil, Harris yeast, iron, "Irradol A," di-calcium phosphate, viosterol and thyroid extract. On admission he was put on a low carbohydrate diet and during the first few months showed considerable improvement. He was found to be allergic to eggs, milk, fish and wheat.

The fever subsided soon after his entrance to the other hospital. Six months later acute joint symptoms recurred accompanied by fever. He complained of pain in the left hip, and x-rays showed it to be subluxated. This attack subsided after several weeks and his condition remained practically unchanged from then until shortly before his re-entry to this hospital. After four years of hospitalization he was allowed to walk about on crutches which he continued to do for a year. At times his appetite was poor and he

frequently lost weight which was regained with difficulty.

Repeated urine examinations showed small amounts of albumin constantly present. Blood examinations showed a persistent polymorphonuclear leukocytosis which varied in degree. At times there was a moderate secondary anemia.

Two months before re-entry to this hospital he began to run a fever, and an x-ray film of the chest suggested bronchopneumonia. A blood culture was sterile, and in a few weeks the febrile attack subsided and an x-ray showed clearing of the pneumonic process. However, one month before re-entry the fever recurred. It was a septic type of fever and persisted up to the time of re-entry. During the week before re-entry he developed bilateral otitis media with mastoiditis on the right.

Physical examination showed a very much debilitated, chronically ill boy. The left eardrum was very thick with a moderate amount of thick pus coming from the middle ear far anteriorly. There was a profuse purulent discharge from the right ear with sagging of the canal wall and definite postaural tenderness and edema. The heart and lungs were negative. There was moderate distention of the abdomen with dilatation of the superficial abdominal veins. The liver was palpable 5 cm. below the costal margin, and the spleen 6 cm. below the costal margin. It was thought that a moderate amount of fluid was present in the abdomen. There was marked wasting of the muscles and subcutaneous tissues of all the extremities, and all the joints of the extremities were very much enlarged. No attempt was made to determine their function.

The temperature was 100.5°F., the pulse 110. The respirations were 25.

The urine examination was negative. The blood showed a red-cell count of 4,000,000 with 50 per cent hemoglobin and a white-cell count of 14,100 with 70 per cent polymorphonuclears. A phenolsulfonephthalein test of renal function showed 50 per cent excretion in three and a quarter hours. The nonprotein nitrogen of the blood was 37 mg. per cent. Staphylococcus albus was cultured from both ears. An x-ray of the mastoids showed destruction of the right tip.

On the day after entry a right simple mastoidectomy was performed. Free pus was encountered in the mastoid cells which gave a pure culture of hemolytic streptococcus. For the next three days his condition was fairly good, but on the eighth hospital day he began to run a septic temperature rising to 101.5° daily, and four days later the daily rise began to reach 104° or 105°. A blood culture on the twelfth day gave no growth. On that day tenderness of the left mastoid was noted. On the following day an examination of the eyes was essentially negative, and no signs of pneumonia could be made out in the lungs. Four days later on the seventeenth day a blood culture gave a profuse growth of hemolytic streptococcus and the white count was 35,800 with 82 per cent polymorphonuclears. On that day it was noted that the left elbow and right knee had increased considerably in size, were generally tender, and obviously contained fluid. Aspiration of both joints yielded thick pus which contained hemolytic streptococci. Sulfanilamide therapy was begun, and three days later a blood culture was negative. However, the patient ran a steadily downhill septic course and died on the twenty-second day.

Presentation of Case

Dr. Margaret Glendy: We know very little about the patient's first illness except that he had a septic fever with swelling of the extremities and abdomen. There is no mention made of the urinary findings, so that in all probability there was no nephritis. The duration was long for deficiency disease, particularly under hospital therapy for four months.

We have on the first admission a two-year-old boy who has had two attacks of septic fever with which there has been enlargement of the hands, feet and abdomen. The type of the enlargement of the abdomen is not clear, but at discharge there was enlargement of the liver. At no time was the spleen felt. There was a generalized lymphadenopathy, a polymorphonuclear leukocytosis and throughout, a negative urine. The x-rays showed soft-tissue thickening with moderate bone atrophy and an abnormally early appearance of centers of ossification. The last two findings would make one believe that this joint change was of fairly long standing and that probably the first illness was of the same nature. Blood cultures, agglutinations, and other studies were negative. No foci of infection were found. Apparently it was a polyarticular non-migratory type of arthritis with septic fever. Incidentally he had been on a markedly deficient diet and, on first admission to the Massachusetts General Hospital, had bulky and malodorous stools which possibly were of the celiac indigestion or sprue type.

Very obviously from the description of the joint, he had Still's disease or rheumatoid arthritis although the spleen was not enlarged. However, in Still's twelve original cases three had no enlargement of the spleen. Still's disease or rheumatoid arthritis in children differs from that in adults in that there is this enlargement of the spleen, and a lymphadenopathy as well as the joint thickening. Felty* has described the same syndrome with arthritis in adults. There were no nodules

*Felty, A. R.: Chronic arthritis in the adult, associated with splenomegaly and leucopenia. Bull. Johns Hopkins Hosp. 35:16-20, 1924.

mentioned in this case, although nodules are found in Still's disease as well as in rheumatic fever. Apparently very little was made of the chest x-ray at the time, but reasoning backward from what we know of Still's disease usually at autopsy there is adhesive polyserositis, and the enlarged heart shadow may have been due to pericarditis. In the ordinary study of the case one of the first things one would think about is nephritis. The swelling of the hands and the feet was not due to edema so that would be ruled out. With the septic fever one would still wonder about septicemia and possibly endocarditis, but there were too many joints involved. They were permanent, did not contain pus or fluid and there was no widening of the joint spaces. The endocarditis problem is still open. There was a tender abdomen. Was the enlargement due to enlarged liver with tenderness and, therefore, perhaps to back pressure from pericarditis with constriction of the great blood vessels, particularly the inferior vena cava? If so, we might have had an x-ray of this type. Certainly the duration of the bronchopneumonia could not account for the enlargement of the right auricle as seen by x-ray. No murmurs were heard, and there was nothing on physical examination in the heart.

"Six months later acute joint symptoms recurred accompanied by fever." That is his third attack with swelling of the joints.

"There was moderate distention of the abdomen with dilatation of the superficial abdominal veins." It looks as if our backward reasoning as to pericardial constriction in the first instance was right and we are now getting some collateral circulation.

The spleen might have been enlarged due to the septic fever.

It says here that the urine examination was negative and yet the urine examination was repeatedly positive in the other hospital. He had been admitted because of an ear condition. Perhaps the otologists were not much interested in the urine.

It is rather interesting that he should have staphylococcus albus in both ears and hemolytic streptococci from the mastoid, and one concludes that the former was probably a contamination from the external auditory canal.

"On the following day examination of the eyes was essentially negative." I imagine that was for choking, although in Still's disease there is frequently exophthalmos. But that would be of little importance in connection with this acute flare-up.

During the interval at the other hospital he had two more acute attacks with progression of the disease in his joints.

His second entry to the hospital was primarily because of bilateral suppurative otitis media with right mastoiditis and, shortly after, with left mastoiditis. Despite the operation the septic temperature persisted and a pure hemolytic streptococcus was found in the mastoid and later in the blood stream. At this time there was a very high white blood count and at least two of the joints became swollen and tender and purulent fluid containing hemolytic streptococci was aspirated from them. The onset was coincident with the increase in the rise of the septic temperature. Sulfanilamide brought about a negative blood culture on at least one occasion, but the condition of the patient progressed steadily downhill, with a septic course throughout.

I feel that in the second admission it was purely coincidental that the same joints became septic which had been arthritic previously. I think that this was probably the septic joint we see in children who have acute mastoids with septic temperature of a rather high degree. I believe the child probably had a lateral sinus thrombosis with metastatic joint abscesses and with possibly more joints involved than were mentioned in the report here. I have seen that happen quite frequently in cases that come to us from the Eye and Ear Infirmary. I think at autopsy that that will be part of the findings, and that the Still's disease will now show enlargement of the spleen which may or may not be due to the septic course. I imagine it is probably part of the picture but I think that concomitantly there will be old pericarditis with thickening of the interlobar septum, some trouble in the peritoneum on the basis of the past findings, and possibly amyloid disease of the liver because of the long persistent albuminuria.

Clinical Discussion

Dr. Harold L. Higgins: In regard to these purulent joints, whenever a person has a positive blood culture the streptococcus is likely to center in the diseased tissue. It occurred to me, with the positive blood culture in this case and the diseased joints of considerable duration, that very likely the seat of the abscesses was in the diseased tissue of the joints. Of course with a lateral sinus thrombosis, the most common place for abscesses to appear is in the joints whether they were previously affected or not.

The child's nutritional condition is interesting. Still's disease is a disease which is known to affect the nodes as well as the joints. Now, was the child's poor nutrition merely the result of a prolonged febrile course and poor appetite or was his poor nutrition due to failure of the mesenteric lymph nodes to function? Perhaps there was some

sort of block there which prevented the proper absorption of fat. I think that is possible. It would be interesting to know the status of the mesenteric lymph nodes in this case. Our diagnosis on the wards was the same as Dr. Glendy's — Still's disease, a mastoiditis with lateral sinus thrombosis and metastatic abscesses to the joints.

Dr. Eli C. Romberg: I saw the child six years ago on the ward when he had been there for about a week. I was very much impressed by the extraordinary septic temperature that he had, from 100 to 105°F., the very irregular pulse, from 100 to 160, and the irregular respirations, from 20 to 60. He did not have any unusual glands and I could find no spleen or any enlargement of the heart. Because of his age, and particularly because of the onset of his original rheumatoid involvement at six months of age, and its tendency to involve the temporomandibular joint, the upper cervical vertebral column, and the phalangeal joints, I considered this as a case of Still's disease with a rheumatoid exacerbation. Ordinarily these joints are not painful except during these exacerbations and in spite of the persistent swelling. We felt that this aspect of the case was a problem for the orthopedist who might be expected to give the patient more comfort by splinting. Empirically I gave him aspirin in large doses after which the interns noted that the patient was more comfortable. We put him on a high vitamin diet and daily colonic irrigations. I also recall that he took beer which was supplied by the house officers.

In regard to the nodules about which Dr. Glendy spoke, it is surprising, in spite of the fact that Still describes it as a common occurrence, that we do not see them commonly in this community in association with rheumatism or chorea. We find isolated ones around the elbows and wrists, yet in Montreal, which is not very far from here, it is not unusual to find many nodules in such cases. The entire scalp may be peppered with subcutaneous nodules varying from the size of peas to small nuts, with others along the ribs and vertebral column and near other joints. We felt that this was the type of rheumatoid arthritis that Still described. The prognosis was poor. He never reached a state of good nutrition. Some get better temporarily, walk about, but eventually come back to their previous state. They progressively get worse and are carried off by some intercurrent infection.

Clinical Diagnoses

Still's disease.
Bilateral otitis media, suppurative acute, with right mastoiditis.
Septicemia.

Dr. Glendy's Diagnoses

Still's disease — with constrictive pericarditis, pleurisy and peritonitis and amyloid disease of liver.
Bilateral otitis media, suppurative acute, with bilateral mastoiditis.
Septicemia with lateral sinus thrombosis and metastatic joint abscesses.

Anatomical Diagnoses

Still's disease.
Septic arthritis.
Mastoiditis.
Lateral sinus thrombosis, right.
Septicemia, hemolytic streptococcus.
Pericarditis, fibrous (adhesive).
Pleuritis, fibrous.
Amyloid degeneration: liver, spleen, kidneys and adrenals.

Pathological Discussion

Dr. Tracy B. Mallory: There was no particular diagnostic problem as to the main diagnosis here. It was a very characteristic case of Still's disease, but there was a chance for speculation as to the complications, and Dr. Glendy seems to have hit most of them. There was, for instance, a markedly adherent pericarditis. There was fluid in both pleurae and in the abdominal cavity. The heart, to our minds, seemed to be normal in size and except for the adhesive pericarditis we could find nothing wrong with it. In contrast the liver, spleen and kidneys were all big and all were a little firmer and paler than normal. Microscopic examination showed extensive amyloid disease. There can be no doubt that this was the source of the persistent albuminuria and edema. The terminal event was bilateral mastoiditis, lateral sinus thrombosis and localized meningitis, with a hemolytic streptococcus septicemia. The blood culture was positive again at autopsy as it had been repeatedly before death. We noticed nothing unusual about the mesenteric lymph nodes.

A number of the joints were carefully examined grossly and histologically by Dr. Granville Bennett in conjunction with Dr. Walter Bauer. They show extensive changes of the type seen in rheumatoid arthritis. There was marked degeneration of the articular cartilage, fibroblastic proliferation with the formation of adhesions, and inflammatory infiltration of the synovia.

Dr. Glendy: One point that concerned me about the first illness was the diarrhea. In a recent report* it was stated that in twelve autopsied cases of Still's disease, seven had diarrhea and had definite involvement of the intestinal nodes.

*Portis, R. B.: Pathology of chronic arthritis of children (Still's Disease). Am. J. Dis. Child. 55:1000-1017, 1938.

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FACTS OR OPINIONS?

THE shifting technics by which data are gathered suggest the changes which overtake the country boy as he becomes a sophisticated metropolitan dweller; he begins to wear trousers instead of pants and wears a hard rather than a soft hat. Whether what he thus covers is greatly changed is another matter. Now the familiar questionnaire calls itself a "survey" and expands its mission from that of asking opinions to that of "finding facts." No self-respecting survey pretends to be based on anything other than fact, and most surveyors are righteous in their insistence that facts alone initiate, propagate and consummate their final conclusions.

For each survey there nevertheless must be a base line; a vantage from which to scrutinize its field. In those nations in which all opinion is triangulated into a uniform system, the choice of this basis is not important, but in democracies where opinions are permitted to vary, the reference point of a survey is of as great importance as its conclusion. The facts themselves may appear in a different light when viewed by different observers, and mighty masses are dwarfed when viewed from lofty heights.

A case in point is the distribution of hospital beds in the United States. The Technical Committee on Medical Care has stated that there are some thirteen hundred counties — over 40 per cent — in the United States that have no registered general hospitals.[1] The Council on Medical Education and Hospitals has stated that there are only thirteen counties no part of which is within thirty miles of a hospital.[2] On a "factual basis" there is nothing incompatible in these two statements, but the one-hundred-fold difference in their implications is sufficient for us to ask both the committee and the council of what importance they think the thirty-mile radius is. To what extent may the advantages of a hospital be felt at the periphery of a thirty-mile circle? What is here asked for is opinion — not fact. The Technical Committee on Medical Care also recommends the addition of 360,000 hospital beds and 500 diagnostic centers for areas now inaccessible to hospitals. Might not some of the cost of these additions be wisely spent to make the rural regions accessible over good roads — thus serving other expressions of social security? Or would the speeding of emergency cases over these improved highways contribute to the accidental hazards of living in the country? Again the answers asked are matters of opinion, not of fact.

In spite of the lack of medical facilities available to the rural residents of this country the United States Public Health Service has recently pointed out that they still enjoy definitely lower rates for most illnesses and especially for fatal illnesses.[3] Will our efforts tend to remove this remaining advantage of living in the country? More than ever opinions are needed; in the present situation it is urgent that they be expressed freely.

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PSYCHIATRIC CLINICS FOR SUICIDAL CASES

THE lack of suitable psychiatric treatment cannot be considered to be a captious criticism of large municipal hospitals, or by any means to imply neglect. The majority has never provided a properly trained staff or space for the adequate care of cases showing mental disturbance in accordance with the minimum requirements for either psychiatric examination or treatment. The large number of suicidal admissions warrant careful attention to the problems and needs of this group. Obviously the majority of these cases are not insane in a legal sense and should not be sent to mental hospitals for observation. There should be definite provision of personnel and of space to carry out a program for dealing with the multitude of psychiatric problems encountered in a large municipal hospital. It is true that these are often subacute, but situations regularly arise which require expert psychiatric diagnosis and treatment. The transfer of sick patients to a mental institution is often dangerous or impossible, and handling them in a general hospital not equipped for their care is likewise complicated and unsatisfactory.

Suicide represents the act of emotional adolescence in many cases. Individuals who have a psychoneurotic make-up may find adequate sublimation in other antisocial pursuits, such as drinking, gambling, sex promiscuity or drug addiction. For all these types, some measure of care is provided when and if they reach extremes incompatible with the interests of the community, although this may be custodial or punitive in its nature and not specifically aimed at eradicating the basic difficulties. The poorly adjusted person who is suicidal deserves appropriate care and consideration. The idea of punishment long ago failed as the proper solution. Adequate preventive measures would definitely help to decrease the number of suicides, and the type of such facilities should depend upon the nature of the community. For a general hospital, the establishment of a psychiatric department or clinic similar to those now operating in the Massachusetts General Hospital and the Bellevue Hospital can ultimately save much more than its cost in preventing mental disease, human suffering and loss of life.

For a municipal hospital like the Boston City Hospital, the establishment of a service to provide a brief psychiatric, diagnostic work-up would yield encouraging results. Its staff could work in close conjunction with the social-service department for essentially simple measures, such as giving help in occupational readjustment, the referring of patients to existing institutions for psychologic and social aid, and assistance in revaluating the problems over which these disturbed personalities have become upset. In some individuals the major conflict may exist on a subconscious level and psychotherapy may necessarily be prolonged over a considerable period. Here the outpatient clinic might function to best advantage. In many cases of attempted suicide the need for educating the family is great. Often the person in greatest need of psychiatric treatment is the marital partner who has *not* attempted suicide. Consultation with a psychiatrically trained staff may be of marked value in certain sexual or psychologic tangles. By means of psychiatric service, innumerable problems may be organized and various adjustments may be favored through relatively short periods of psychotherapy.

Financial, vocational and emotional difficulties in themselves can be very disturbing, but often can be ironed out by intelligent advice and guidance. Large sums of money are available for experiment in the causes and prevention of physical disease. The prevention of a disease as great as suicide, which kills more persons than the combined efforts of twenty common medical illnesses, warrants the expenditure of money and energy sufficient to establish and maintain preventive wards, at least for a trial period. There is every reason to believe that such a service in municipal hospitals, such as the Boston City Hospital, would prove useful and economical and, within a short period, become established as a permanent necessity.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
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SPONTANEOUS RUPTURE DURING DELIVERY

Mrs. G. J., a twenty-four-year-old para II, at term, was seen in consultation at home August 15, 1936, twelve hours after a normal delivery.

The patient's physician stated that after fourteen hours of good labor her pains became weaker and irregular, with the cervix dilated four fingers and the presenting part not engaged. He did not state when the membranes had ruptured. Because of the irregularity of the pains, 0.5 cc. of pituitary extract was administered. This resulted in spontaneous delivery after three or four strong uterine contractions. He further stated that there was a good deal of bleeding after the delivery of the placenta, at which time the patient herself became very pale and her pulse rapid and thready. Because of this collapse, the patient was transfused with 500 cc. of citrated blood four hours after the delivery of the placenta. The improvement following the transfusion was temporary, and because of the increasing pallor and the rapidity of her pulse, the consultant was called.

The family history was not important. The patient had undergone no serious illnesses or operations. Catamenia had begun at thirteen, were regular with a thirty-day interval, and lasted five days. Her last period was November 8, making her expected date of confinement August 15. Her first pregnancy was normal; her present pregnancy was normal up to the time of delivery.

On physical examination the patient was well-developed and nourished with marked pallor of the skin and mucous membranes. The lungs were normal throughout; there were no rales. The heart was not enlarged; there was a systolic murmur at the base. The pulse was 124, the temperature 98°F., and the blood pressure 94 systolic, 48 diastolic. The fundus could be felt two fingers above the umbilicus and seemed drawn to the left. There was considerable tenderness in the left lower quadrant and flank. There was a moderate bloody, vaginal discharge, greater than the normal flow of lochia. The patient was transferred to the hospital without having had a vaginal examination.

After admission to the hospital a vaginal examination revealed a laceration of the cervix on the left extending up as far as the finger could reach. The patient was again transfused with 500 cc. of citrated blood. A diagnosis of lower uterine rupture was made, and operation was immediately performed.

Under ether anesthesia the abdomen was opened in the median line. There was no free blood in the abdominal cavity. The fundus was mottled, and a large hematoma distended the left broad ligament. The uterine rupture had occurred between the leaves of the broad ligament, so that there was no intraperitoneal hemorrhage. A panhysterectomy and left salpingo-oophorectomy were performed. The abdomen was drained through the vagina by iodoform gauze. Examination of the uterus revealed a rupture extending from the external os up the left side almost to the left cornu. The patient's condition following the operation was definitely improved. The pulse had dropped to 98 and was of better quality.

Her convalescence was uneventful save for pyelitis which developed on the fifth day and was treated by bladder irrigation and 20 gr. of methenamine. She was discharged on the fifteenth day.

Comment. The use of pituitary extract when the head is not engaged is always open to question. The irregularity of labor suggests the formation of a contraction ring. The history does not tell when the membranes ruptured. Undoubtedly the contractions following the use of pituitary extract were so strong that the rupture followed.

The value of transfusion is well illustrated in this particular case. Of course, the patient was matched before transfusion was done, although this detail is not in the history. The removal of the patient to the hospital before vaginal examination was performed was also a wise step. The diagnosis was made definite by a later vaginal examination, and the tear thus revealed was one that could not have been adequately treated by cervical ligatures. Laparotomy was the only procedure which would adequately meet this type of rupture, and hysterectomy was the only operation.

A series of selected case histories by members of the section will be published weekly.

Comments and questions by subscribers are solicited and will be discussed by members of the section.

DEATHS

BRENNAN — EDWARD F. BRENNAN, M.D., of Guild Square, Norwood, died September 9. He was in his fifty-fifth year.

Born in Natick he graduated from the Natick high school and received his degree from the Tufts College Medical School in 1908. Dr. Brennan had practiced for more than twenty-five years in Norwood.

He was a member of the American Medical Association and the Massachusetts Medical Society.

His widow survives him.

BYCHOWER — VICTOR BYCHOWER, M.D., of 226 Cross Street, Malden, died at Bethlehem, New Hampshire, September 2. He was in his seventy-eighth year.

Dr. Bychower received his degree from the Kaiser-Wilhelms-Universität Medizinische Fakultät in 1888 and practiced in Malden for forty years. He was a member of the American Medical Association and the Massachusetts Medical Society.

His widow survives him.

PACKARD — EDWARD A. PACKARD, M.D., of Waban, died at South Harpswell, Maine, September 8. He was in his eighty-first year.

Born at Auburn, Maine, he graduated from Bowdoin College in 1883 and received his degree from Columbia University College of Physicians and Surgeons in 1886.

Dr. Packard served his internship in New York and practiced in that city and Worcester before coming to Boston. Twenty years ago he retired from practice. He was a member of the American Medical Association and the Massachusetts Medical Society.

His sister and a son survive him.

MISCELLANY

NOTE

Dr. Elliott P. Joslin, emeritus professor of clinical medicine at the Harvard Medical School, delivered the two Malthe lectures and conducted a clinic in Oslo on September 5, 6 and 7; he will participate in a symposium on protamine insulin with Dr. H. C. Hagedorn, of Copenhagen, and Dr. Wilhelm Falta, of Vienna, before the Gesellschaft für Verdauungs- und Stoffwechsel-Krankheiten in Stuttgart on September 23. — *Science.*

CORRESPONDENCE

NEW REGULATIONS OF THE MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

To the Editor: This department now has authority, granted by a recent act of the legislature, to adopt rules and regulations with regard to the control of diseases dangerous to the public health; such regulations have been recently adopted. I should like to have you print these regulations for the information of physicians.

ALTON S. POPE, M.D.,
Deputy Commissioner of Public Health,
Department of Public Health.

State House,
Boston.

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RULES AND REGULATIONS OF THE DEPARTMENT OF PUBLIC HEALTH RELATIVE TO DISEASES DANGEROUS TO THE PUBLIC HEALTH, MADE UNDER THE PROVISIONS OF SECTION 6, CHAPTER 111, OF THE GENERAL LAWS, AS AMENDED BY CHAPTER 265 OF THE ACTS OF 1938

Regulation 1. Reporting cases of diseases dangerous to the public health on dairy farms. When a case of typhoid fever, paratyphoid fever, diphtheria, scarlet fever, epidemic or septic sore throat, poliomyelitis (acute anterior), amebic or bacillary dysentery, or Asiatic cholera occurs on any farm or dairy producing milk, cream, butter or other dairy products for sale, it shall be the duty of the physician in attendance to report immediately to the local board of health the existence on such farm or dairy of such case. If no physician is in attendance it shall be the duty of the owner or person in charge of such farm or dairy to report forthwith to the local board of health the name and address of any person, who is affected with a disease presumably dangerous to the public health, and who is employed or resides on or in such farm or dairy or who comes in contact in any way therewith or with its products.

It shall be the duty of the local board of health to report immediately to the state district health officer by telephone or telegram the existence on such farm or dairy of a case of disease mentioned in this regulation, together with all facts as to the isolation of such case, and to give the names of the cities and towns to which such dairy products are delivered.

Regulation 2. Reporting of illness suspected of being due to consumption of food. Every physician and every superintendent or other person in charge of any school, hospital, institution, dispensary, laboratory, labor camp or other camp, who shall have knowledge of the occurrence of a number or group of cases of illness believed to have been due to the consumption of food, shall report the same immediately, by telephone or telegram, to the local board of health.

Regulation 3. Notification of outbreaks of food poisoning, diarrhea, jaundice, epidemic influenza, glandular fever, sore throat and undiagnosed febrile disease. Whenever there shall occur in any municipality an outbreak of suspected food poisoning or an unusual prevalence of diarrhea, gastroenteritis, enteritis, colitis, enterocolitis, cholera nostras, cholera infantum or other disease in which diarrhea is a prominent symptom, or whenever jaundice, epidemic influenza, glandular fever, sore throat or any undiagnosed febrile disease is unusually prevalent, it shall be the duty of the health officer to report immediately the existence of such an outbreak to the Department of Public Health by telephone or telegram.

Regulation 4. Control of carriers. No person found to be harboring typhoid, paratyphoid, dysentery or other pathogenic intestinal organisms may engage in a food-handling capacity until it is demonstrated to the satisfaction of the Department of Public Health that the said person no longer harbors the organism.

Regulation 5. Carrier, contact, isolation, placard and quarantine are defined as follows:

a. Carrier. A person who, without symptoms of a disease dangerous to the public health, harbors and may disseminate the specific micro-organisms of that disease.

b. Contact. Any person known to have been sufficiently near an infected person or animal to have been presumably exposed to transfer of infectious material directly, or by articles freshly soiled with such material.

c. Isolation. The separation of persons suffering from any disease dangerous to the public health, or carriers of the infecting micro-organism, from other persons, in such places and under such conditions as will prevent the direct or indirect conveyance of the infectious agent to susceptible persons.

d. Placard. An official notice, written or printed, posted as a warning of the presence of a disease dangerous to the public health on the premises or in the apartment or room so placarded.

e. Quarantine. The restriction to the premises, house or apartment of materials and persons that presumably have been exposed to a disease dangerous to the public health.

Regulation 6. Isolation and quarantine requirements. The following shall be the minimum requirements for isolation and quarantine of diseases dangerous to the public health:

Isolation and Quarantine Requirements by the Massachusetts Department of Public Health

Disease	Minimum Period of Isolation of Patient	Minimum Period of Quarantine of Contacts			Placard
		adults	immune children	children not immune	
Actinomycosis	No restrictions	No restrictions	No restrictions	No restrictions	No
Anterior poliomyelitis (infantile paralysis)	Two weeks from onset of disease, and thereafter until acute symptoms have subsided.	Notes 1 and 2	Until two weeks have elapsed from date of last exposure.	Until two weeks have elapsed from date of last exposure.	Yes
Anthrax	Until lesions are healed	No restrictions	No restrictions	No restrictions	No
Asiatic cholera	Same as typhoid fever	Seven days from last exposure, and until a negative stool is obtained. Note 3.	Same as adult	Same as adult	Yes
Chickenpox	One week from appearance of eruption and thereafter until all crusts have disappeared.	No restrictions	No restrictions	No restrictions	No
Cholecystitis of typhoid origin (typhoid carrier). See Regulation 4.	Supervision by local board of health until released from the carrier list by the Department of Public Health.	Food handlers living in a household with a typhoid carrier shall be excluded from their occupations unless they have been inoculated with typhoid vaccine within two years and agree to observe precautions prescribed by the board of health.	No restrictions	No restrictions	No
Diphtheria	One week from date of onset and thereafter until two successive negative cultures, taken at least twenty-four hours apart, from both nose and throat, have been obtained.	Food handlers and persons, whose occupation brings them into contact with children living in a family in which a case exists, shall be subject to the same restrictions as children only in so far as it applies to their occupation.	If immune as shown by Schick test or on the basis of a previous attack of the disease, no restrictions provided they live away from home or the case is hospitalized, and if two consecutive negative nose and throat cultures taken at an interval of not less than twenty-four hours have been obtained.	Until child lives away from home one week and until two negative nose and throat cultures taken at an interval of not less than twenty-four hours have been obtained. No restrictions thereafter if child continues to live away from home.	Yes
Dog bite	No restrictions	No restrictions	No restrictions	No restrictions	No
Dysentery, amebic	Same as typhoid fever	Note 3	No restrictions	No restrictions	No
Dysentery, bacillary	Same as typhoid fever	Note 3	No restrictions	No restrictions	No
Encephalitis lethargica	One week after onset	No restrictions	No restrictions	No restrictions	No
German measles	Four days from appearance of rash	No restrictions	No restrictions	No restrictions	No
Glanders	Until lesions are healed.	No restrictions	No restrictions	No restrictions	No
Gonorrhea	See "Regulations Governing the Control of Gonorrhea and Syphilis."				
Hookworm disease	No restrictions	No restrictions	No restrictions	No restrictions	No
Leprosy	Until disease is arrested.	No restrictions	No restrictions	No restrictions	No
Lobar pneumonia	Until recovery	No restrictions	No restrictions	No restrictions	No
Malaria	No restrictions	No restrictions	No restrictions	No restrictions	No
Measles	One week from appearance of rash	Note 1	No restrictions. Note 4.	Exclusion from school for fourteen days from date of last exposure. Note 7.	No
Meningococcus meningitis	Two weeks from onset of disease, and thereafter until all acute symptoms have subsided.	Notes 1 and 2	Until ten days from date of last exposure	Until ten days from date of last exposure	Yes
Mumps	One week from onset of disease, and thereafter until all swelling of salivary glands has disappeared.	No restrictions	No restrictions	No restrictions	No
Ophthalmia neonatorum	One week after subsidence of symptoms and until two successive cultures from each eye at an interval of not less than forty-eight hours are negative for gonococci.	No restrictions	No restrictions	No restrictions	No

Isolation and Quarantine Requirements by the Massachusetts Department of Public Health
(Continued)

Disease	Minimum Period of Isolation of Patient	Minimum Period of Quarantine of Contacts			Placard
		adults	immune children	children not immune	
Paratyphoid fever, A and B	Same as typhoid fever	Note 3	No restrictions	No restrictions	No
Pellagra	No restrictions	No restrictions	No restrictions	No restrictions	No
Pfeiffer-bacillus meningitis	No restrictions	No restrictions	No restrictions	No restrictions	No
Plague	One week after subsidence of all symptoms	In pneumonic cases, until seven days, and no restrictions thereafter provided patients are hospitalized or they live away from home.	Same as adults	Same as adults	Yes
Psittacosis	Until recovery	No restrictions	No restrictions	No restrictions	No
Rabies	During course of disease	No restrictions	No restrictions	No restrictions	No
Scarlet fever	Uncomplicated Cases: three weeks from date of appearance of rash. Careful attention should be given to nose and throat to detect existence of discharge or inflammation before considering case as uncomplicated. (If upper respiratory tract symptoms appear during month after release from isolation, precautions should be re-established.) Complicated Cases: four weeks and thereafter until abnormal discharges shall have ceased, swollen glands subsided, or three successive cultures of abnormal discharge shall have been found free of hemolytic streptococci.	Food handlers and persons, whose occupation brings them into contact with children, living in a family in which a case exists, shall be subject to the same restrictions as children only in so far as it applies to their occupation.	If immune as shown by Dick test or on the basis of a previous attack of the disease, no restrictions, provided they live away from home, or cases are hospitalized.	Until child lives away from home one week; no restrictions thereafter, if child continues to live away from home.	Yes
Rocky Mountain spotted fever	No restrictions	No restrictions	No restrictions	No restrictions	No
Septic sore throat	Until one week after onset and until recovery, except milk handlers, who shall be excluded from their occupation until satisfactory evidence is obtained that the danger of conveying the disease has passed.	No restrictions except for milk handlers	Same as for adults	Same as for adults	No
Smallpox	Three weeks from onset of disease and thereafter until all crusts have disappeared and skin has healed.	Note 5	Note 5	Note 5	Yes
Suppurative conjunctivitis (acute epidemic conjunctivitis, pink eye)	Exclusion from school and public gatherings until recovery	No restrictions unless suspected of being infected.	Same as for adults	Same as for adults	No
Syphilis	See "Regulations Governing the Control of Gonorrhea and Syphilis."				
Tetanus	No restrictions	No restrictions	No restrictions	No restrictions	No
Trachoma	Exclusion from general school classes during acute stage	No restrictions	No restrictions	No restrictions	No
Trichinosis	No restrictions	No restrictions	No restrictions	No restrictions	No
Tuberculosis (all forms)	Note 6	No restrictions	No restrictions	No restrictions	No
Tularemia	During acute stage	No restrictions	No restrictions	No restrictions	No
Typhoid fever. (Typhoid carrier: see "Cholecystitis of typhoid origin.")	One week after subsidence of clinical symptoms. Thereafter may be released on special permission of and under the supervision of the local board of health, supervision to continue until three successive negative stool and urine cultures, secured at intervals of at least one week apart, have been obtained.	Note 3	No restrictions	No restrictions	No

Isolation and Quarantine Requirements by the Massachusetts Department of Public Health
(Concluded)

Disease	Minimum Period of Isolation of Patient	Minimum Period of Quarantine of Contacts			Placard
		adults	immune children	children not immune	
Typhus fever	In vermin-free room until recovery	In presence of lice, until fourteen days after last exposure	Same as for adults	Same as for adults	No
Undulant fever	No restrictions	No restrictions	No restrictions	No restrictions	No
Whooping cough	Three weeks from beginning of spasmodic cough	Note 1	No restrictions. Note 4.	Until two weeks from last exposure	No
Yellow fever	In mosquito-proof room first four days of fever	No restrictions	No restrictions	No restrictions	No

NOTES

1. Schoolteachers, only as it applies to their school activities, shall be subject to the same restrictions as children.

2. Food handlers and persons whose occupation brings them in contact with children have no restrictions if they live away from home.

3. Food handlers living in a family where a case of typhoid, cholera, amebic or bacillary dysentery or paratyphoid A or B exists shall be excluded from their occupation so long as they continue to live in the same house in which the case exists, and thereafter until freedom from infection, as judged by clinical and laboratory evidence, has been demonstrated to the satisfaction of the Department of Public Health.

4. A child shall be considered as having had the disease if so shown by the record of the local board of health or by a sworn statement from the parent or guardian that the child has had the disease elsewhere.

5. Contacts shall be quarantined until three weeks have elapsed from the date of last exposure unless immunized by a previous attack, by a recent successful vaccination, or showing the immune reaction.

6. Patients with open tuberculosis should in most cases receive sanatorium treatment. Those who remain in their homes shall observe all precautions necessary to prevent infection of the members of their families and of others with whom they may come in contact. This shall include approved methods of collection and disposal of the sputum, the sterilization of any articles of clothing and of toilet articles which may become contaminated by the sputum, the use of separate dishes and eating utensils and proper sterilization of the same. The patient should sleep in a separate room and must in any case occupy a separate bed. For further details concerning precautions in home care see *Home Care of Tuberculosis Patients*, a pamphlet of the Department of Public Health.

No person who has tubercle bacilli in the sputum or other bodily discharges shall be allowed to engage in the preparation or serving of food or be employed on a dairy farm or in a dairy plant in any capacity which may bring him in contact with milk or milk products.

7. In communities with good school inspection children exposed to measles may be permitted to attend school seven days after the first exposure, thereafter to be excluded from the seventh to fourteenth days.

—Approved by Massachusetts Department of Public Health, August 9, 1938.

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Regulations of the Department of Public Health Relative to the Control of Gonorrhea and Syphilis, Made Under the Provisions of Section 6, Chapter 111, of the General Laws, as Amended by Chapter 265 of the Acts of 1938

Reporting

1. Whenever a physician has reason to believe that a person whom he has examined is suffering from gonorrhea or syphilis in any form or stage, he shall: —

a. Report immediately to the Department of Public Health, on forms provided by the department for that purpose, stating the name of the disease and its form or stage, the age, sex, marital status and community of residence of said patient and whether or not said patient has been under treatment elsewhere for the same infection, but not the patient's name or street address except as hereinafter provided.

b. Report the change in medical adviser to the physician, if any, who last treated said patient for the infection.

c. Report to the department any failure on the part of said patient to return for observation or treatment, unless said physician has satisfactory evidence that said patient is under the care of another physician for the infection, or has received adequate treatment for the infection, stating the name, address, age, sex and marital status of said patient, the name and stage or form of the disease, the date of the last visit to said physician, and, in case of gonorrhea, or syphilis of less than two years' duration, the approximate date of infection. Said report shall be made to the department not more than one week after the date of the missed appointment in case of syphilis of less than two years' duration (whether acquired or congenital), syphilis in a pregnant woman and gonorrhea in any form or stage; and not more than two weeks after the date of the missed appointment in all other forms or stages of syphilis, provided that any physician who has available and uses the services of a competent epidemiologist within the time limits herebefore prescribed, may defer reporting said patient's failure to return for observation or treatment for two weeks from the date of the missed appointment where one week is herebefore prescribed and for four weeks where two weeks are herebefore prescribed.

d. Report to the department the name, address, sex, age, marital status and occupation of the patient who, having syphilis, has open lesions of the disease in the mouth or upon exposed portions of the body unless said physician is satisfied that said patient has discontinued his occupation, or is so employed that no other person is likely to be exposed to said patient's infection.

e. Report to the department the patient who fails to co-operate with said physician in his attempt to bring to medical observation those persons, including members of the patient's family, from whom said patient may have acquired, or to whom said patient may have transmitted, his infection, stating the name, address, age, sex and marital status of said patient and the reason for the report.

f. Report to the department the name, address, age, sex and marital status, or as complete identification as can be obtained, of any person, other than the patient's husband or wife, from whom a patient who has gonorrhea, or syphilis of less than two years' duration, may have acquired, or to whom he may have transmitted, his infection; provided that if said physician is satisfied that such person cannot be identified by said patient or, within two weeks of identification to said physician has been brought under medical observation, such report need not be made.

2. The department may forward to any board of health of any city or town a report stating the name, address or place frequented by or place of employment of, age, sex and marital status of any person reported to the department by name under Regulation 1, together with the reason for the report. Immediately upon the conclusion of such action as said board of health may take following receipt of such report, the said board of health shall report the result of such action to the department, provided that if said person cannot be found, the said board shall so report to the department.

CONTROL

3. Whenever a board of health shall receive a report from the department stating the name and address of, or place frequented by, a person who resides, or may be found, in the community in which said board has jurisdiction, and who is alleged to have been exposed to infection with gonorrhea or syphilis, the said board shall immediately advise said person to obtain medical attention. Not less than five days or more than one week after so advising said person, the said board shall determine whether, and by whom, such examination has been made. If, within two weeks of the date when first so advised, said person has not satisfied said board that he is under medical care or is free from infection with gonorrhea or syphilis, the said board shall isolate said person until said board is satisfied that said person is not infected with gonorrhea or syphilis.

4. Whenever a board of health shall receive a report from the department stating the name and address of, or place frequented by, a person who resides, or may be found, in the community in which said board has jurisdiction, and who has prematurely discontinued treatment for gonorrhea or syphilis, the said board shall immediately request said person to return to medical care. Not less than five days or more than one week after such request, the said board shall determine whether, and with whom, medical attention has been resumed. If, within two weeks of the date of said request, the said person has not resumed medical attention, the said board shall isolate said person until it has been satisfied that said person's infection with gonorrhea or syphilis is no longer communicable, or that said person will immediately return to medical care for said infection.

5. Whenever a board of health shall receive a report from the department stating the name and address or place frequented by, a person who resides, or may be found, in the community in which said board has jurisdiction, and who has open lesions of syphilis in the mouth or upon exposed portions of the body and who is employed at any occupation requiring contact with other persons, the said board shall request said person to discontinue such occupation until the said board is satisfied that the lesions are healed and that the said person will remain under medical care for the infection. If the said person fails to discontinue such occupation immediately, the said board shall remove said person to his home or to a hospital where he shall be isolated until said board is satisfied that the lesions are healed and that said person will remain under medical care for the infection.

6. Whenever a board of health shall receive a report from the department stating the name and address or place frequented by a patient suffering from gonorrhea, or syphilis of less than two years' duration, who resides or may be found in the community in which said board has jurisdiction, and who is said to have failed to co-operate with his physician in said physician's attempt to bring to medical observation any person from whom said patient may have acquired, or to whom he may have transmitted, said infection, said board shall immediately isolate said patient and shall retain him in isolation until he will so co-operate, or until his infection is no longer communicable.

—Approved by the Massachusetts Department of Public Health, August 9, 1938.

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Regulations of the Department of Public Health Relative to the Conveyance of Bodies Dead of any Disease Dangerous to the Public Health, Made Under the Provisions of Section 107, Chapter 111, of the General Laws, as Amended by Chapter 265 of the Acts of 1938

The following terms hereinafter used are defined as follows:

"To transport" shall mean to convey or remove a dead human body from the place of death.

"To ship" shall mean to convey a dead human body by train, boat or airplane.

Rule 1. No bodies dead of any disease dangerous to the public health may be transported or shipped, except as provided by Rules 3 and 4, without having been prepared for burial by thorough embalming and disinfecting.

Rule 2. Bodies dead of anthrax, glanders, leprosy, plague, smallpox or tularemia, which may produce dangerous surface lesions, and if such lesions occur on such bodies, may not be transported unless further prepared by being enveloped in a sheet saturated with bichloride of mercury, 1:500, and securely pinned; provided that if such lesions do not occur on head, neck or hands such parts may not necessarily be enveloped. When in doubt as to the preparation of such bodies for transportation, consult with the representative of the local board of health.

Rule 3. Bodies dead of diseases dangerous to the public health but without dangerous surface lesions may be transported to the morgue of a funeral director without

arterial and cavity embalming provided that such bodies have been prepared before transportation by thorough surface washing and disinfecting and by stopping and disinfecting of orifices and by enveloping in a sheet saturated with 1:500 bichloride of mercury and securely pinning the same. Before removal from such morgue the body must be thoroughly embalmed and disinfected.

Rule 4. Bodies dead of any disease dangerous to the public health may be transported directly to the place of burial within twenty-four hours of death without arterial and cavity embalming, provided such bodies are prepared before transportation by thorough surface washing and disinfecting and by the stopping and disinfecting of orifices and by enveloping in a sheet saturated with 1:500 bichloride of mercury and securely pinning the same. Such unembalmed bodies are to be placed in sealed caskets or cases not to be reopened, and may be shown only under sealed glass panels. If such unembalmed bodies are to be shipped, they must be placed in hermetically sealed cases.

Rule 5. Bodies dead of any disease dangerous to the public health shall not be accompanied by articles which have been exposed to the infection of the disease, unless such articles have been properly disinfected.

Rule 6. No disinterment of the bodies of any persons who have died of any disease dangerous to the public health shall be allowed within six months from date of death, except by special permission of the representative of the local board of health.

—Approved by the Massachusetts Department of Public Health, August 9, 1938.

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REGULATIONS OF THE DEPARTMENT OF PUBLIC HEALTH RELATIVE TO FUNERALS OF PERSONS DEAD OF ANY DISEASE DANGEROUS TO THE PUBLIC HEALTH, MADE UNDER THE PROVISIONS OF SECTION 107, CHAPTER 111, OF THE GENERAL LAWS, AS AMENDED BY CHAPTER 265 OF THE ACTS OF 1938

Rule 1. Funerals of those dead of anterior poliomyelitis, Asiatic cholera, diphtheria, encephalitis lethargica, leprosy, meningococcus meningitis, plague, psittacosis, scarlet fever, smallpox, streptococcus sore throat or typhus fever, when conducted on the premises where such deceased persons died, shall be attended only by members of the immediate household, clergymen, undertaker and undertaker's assistants; when held from a place other than where such persons died, the representative of the local board of health may permit a public funeral if the bodies, in his opinion, have been prepared or enclosed according to the regulations of the Department of Public Health and provided such persons as are probable carriers of the infection, by reason of conduct, shall be forbidden to attend such funeral.

Rule 2. Bodies dead of anthrax, glanders, leprosy, plague, smallpox or tularemia, which may produce dangerous surface lesions, and if such lesions occur on such bodies, after preparation for transportation as regulated by the department, must be placed before the funeral in a sealed casket, not to be reopened, and the funeral may be either public or private. Such bodies may be shown only under a sealed glass-paneled burial case.

—Approved by the Massachusetts Department of Public Health, August 9, 1938.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

To the Editor: In addition to the articles enumerated in our letter of July 9 the following have been accepted:

Abbott Laboratories

Cevitamic Acid — Abbott
Tablets Cevitamic Acid — Abbott, 25 mg.
Tablets Cevitamic Acid — Abbott, 100 mg.

Drug Products Co., Inc.

Pulvoids Digitalis Folium, ½ gr.
Pulvoids Digitalis Folium, ¾ gr.
Pulvoids Digitalis Folium, 1½ gr.

Jensen-Salsbery Laboratories, Inc.

Diphtheria Toxoid, Alum Precipitated (Refined), one 10 cc. vial package

Sharp & Dohme

Propadrine Hydrochloride Capsules, ¾ gr.
Propadrine Hydrochloride Solution 3 per cent

E. R. Squibb & Sons

Refined Tetanus Toxoid, Alum Precipitated— Squibb, two 1 cc. vials package

United States Standard Products Co.

Polyanaerobic Antitoxin (Tetanus-Gas-Gangrene) Refined and Concentrated, one vial package

PAUL NICHOLAS LEECH, *Secretary.*

535 North Dearborn Street,
Chicago, Illinois

NOTICES

ANNOUNCEMENTS

ALBERT E. ROGERS, M.D., announces the removal of his office to 226 Bay State Road (corner of Granby Street), Boston.

M. LEOPOLD BRODNY, M.D., announces the removal of his offices to 520 Beacon Street, Boston. Telephone Kenmore 3200.

CHARLES F. FERGUSON, M.D., announces the opening of an office at 4 Abbott Road, Wellesley Hills.

CONFERENCE ON RURAL MEDICINE

The program for the Conference on Rural Medicine to be held at the Mary Imogene Bassett Hospital, Cooperstown, New York, is as follows:

FRIDAY, OCTOBER 7

Morning Session, 9:30 a. m.

PART I. RURAL MORBIDITY

Chairman: To be announced.

1. Introductory Remarks. G. M. Mackenzie, The Mary Imogene Bassett Hospital.
2. Vital Statistics of Otsego County, New York. Joseph V. DePorte, Director, Division of Vital Statistics, State of New York Department of Health.
3. Statistical Analysis of 15,000 Consecutive Admissions to a Rural Hospital in Otsego County, New York. Edwin L. Crosby, Department of Biostatistics, School of Hygiene and Public Health, Johns Hopkins University, Baltimore.
4. Emergency Abdominal Surgery in a Rural Hospital. Monroe A. McIver, The Mary Imogene Bassett Hospital.
5. Cardiac Disease in a Rural Community. Francis F. Harrison, The Mary Imogene Bassett Hospital.

6. Vascular Disease in a Rural Community. David Kydd, The Mary Imogene Bassett Hospital.

Discussion opened by Louis I. Dublin, third vice-president and statistician, Metropolitan Life Insurance Company, New York City.

Afternoon Session, 2:15 p. m.

PART II. HEALTH DEPARTMENT PROGRAMS AND SCHOOL HEALTH PROGRAMS IN RURAL AREAS

Chairman: Edward S. Godfrey, Jr., commissioner of health, State of New York Department of Health.

1. Needs and Opportunities of the Rural Health Department. Allen W. Freeman, professor of public-health administration, School of Public Health and Hygiene, Johns Hopkins University, Baltimore.
2. Evaluation of Routine School Health Examinations. Marjorie F. Murray, The Mary Imogene Bassett Hospital.
3. Medical Problems of School Health Supervision. Haven Emerson, director, De Lamar Institute of Public Health, Columbia University, College of Physicians and Surgeons, New York City.

Discussion by Harry S. Mustard, professor of preventive medicine, New York University College of Medicine, New York City, and Gilbert deL. Forbes, Kendall, New York.

PART III. POSTGRADUATE MEDICAL EDUCATION IN RURAL AREAS

Chairman: Thomas P. Farmer, chairman, Council Committee on Public Health and Education of the Medical Society of the State of New York.

1. Postgraduate Medical Education: The Michigan experience. James D. Bruce, University of Michigan, vice-president in charge of university relations.
2. Postgraduate Training for Practitioners in Rural Areas: The needs and the methods. John B. Youmans, associate professor of medicine, Vanderbilt University, Nashville.

Discussion by Dr. Samuel H. Proger.

SATURDAY, OCTOBER 8

Morning Session, 9:30 a. m.

PART IV. ECONOMICS OF RURAL MEDICINE

Chairman: Owen D. Young, chairman of the board, General Electric Company.

1. Hospitals and Hospitalization in Rural Communities. Vane M. Hoge, Division of Public Health Methods, United States Public Health Service.
2. The Economics of Rural Medicine from the Viewpoint of the Country Doctor. Lloyd C. Warren, president, Otsego County Medical Society, Franklin, New York.
3. Rural Medicine and Economics. Charles C. McCoy, The Mary Imogene Bassett Hospital.
4. Economics of Rural Medicine. Michael M. Davis, chairman, Committee on Research in Medical Economics, New York City.
5. Medical Care for Rural America. Rosco G. Leland, director, Bureau of Medical Economics, American Medical Association, Chicago.

Discussion by Robert A. Polson, extension rural sociologist, New York State College of Agriculture, Cornell University, Ithaca.

CLINICAL CONGRESS OF THE CONNECTICUT STATE MEDICAL SOCIETY

Each successive year the Clinical Congress has gained in importance and size. Judging from the early registration returns, this year will find it larger than ever before. Its sessions, September 20, 21 and 22, will probably surpass last year's attendance record of 700. The registration fee for the Congress is $2.00. Medical students, interns and residents of the local medical school and hospitals will be welcomed without charge.

All morning sessions, which will be held at the auditorium in the Sterling Law Buildings, will begin promptly at 9:30 a. m. Afternoon sessions will be held in the buildings of the New Haven Hospital and Yale School of Medicine and will begin promptly at 2:15 p. m. Evening meetings, which have been arranged by the various Sections of the Society, will likewise be held at New Haven Hospital or Yale University School of Medicine at 8:15 p. m. and will be open to all members of the Congress.

On Tuesday morning, September 20, Dean Stanhope Bayne-Jones of the Yale School of Medicine will preside. Dr. Nicholson J. Eastman, professor of obstetrics, Johns Hopkins University School of Medicine, will speak on "Pyelitis in Pregnancy"; Dr. Edward D. Churchill, professor of surgery at Harvard Medical School, will speak on "Surgery in Pulmonary Disease"; Dr. Harry Goldblatt, professor of experimental pathology, Western Reserve University, will speak on "Pathogenesis and Treatment of Hypertension" and the series of five-minute talks will be presented by Drs. Preu, German, Thompson, Foster and Kirschbaum. In the afternoon a bedside clinic will be given by Dr. Churchill. Panel discussions will include "Pyelitis in Pregnancy" by Drs. Morse, Eastman, Deming, Howard, Lavietes and Perrins; "Surgery in Pulmonary Disease," by Drs. Lindskog, Churchill, Beecher, Campbell, Canfield, Lynch, Soper, Standish and Urquhart; "Pathogenesis and Treatment of Hypertension," by Drs. Peters, Goldblatt, Ayman, Blumgart, Glenn and Weiss. At 4:00 p. m. Dr. Foster will show motion pictures to the Section on Obstetrics and Gynecology. At 8:15, Dr. Ogden presiding, the Section on Radiology will hear Dr. Grantley Taylor, of Boston, speak on "Management of Regional Lymph Nodes in the Treatment of Carcinoma." At 8:15 the Section on Anesthesia meeting with the American Society of Anesthetists with Dr. Sword presiding will hear papers by Dr. Mario Garofalo, of Hartford, on the "Present Status of Anesthesia with Pentothal Sodium"; Dr. F. A. D. Alexander, of Albany, on "Reorganization of the Department of Anesthesia at the Albany Hospital" and Dr. R. Charles Adams, of Rochester, Minnesota, on "Methods of Anesthesia for Operations About the Face and Neck."

On Wednesday morning Dr. Hugh Campbell, president of the Connecticut State Medical Society, will preside. Dr. Henry T. Chickering, assistant professor of clinical medicine, Columbia University, will speak on "The Treatment of Pneumonia"; Dr. Cornelius P. Rhoads, associate member of the Rockefeller Institute for Medical Research, will speak on "The Role of Vitamins in Health and Disease" and Dr. Samuel T. Orton, former professor of neurology, Columbia University, will speak on "Disorders in Speech, Reading and Writing in Children." Five-minute talks will be given by Drs. Bayne-Jones, Deming, Tileston, White and Goldstein. In the afternoon there will be panel discussions on the "Treatment of Pneumonia" by Drs. Blake, Chickering, Finland, MacLeod, Rogers, Welch and Wentworth; "The Role of Vitamins in Health and Disease" by Drs. Lavietes, Rhoads, Aub, Hecht, Ralli and Weiss; "Disorders in Speech, Reading and Writing in Children" by

Drs. Kahn, Orton, Cunningham, Gesell, Ilg and Salinger. In the evening at 8:15 Dr. James D. Gold will preside at the meeting of the Section on Dermatology and Syphilology and Dr. Isadore Rosen, of New York City, will speak on "Mucous Membrane Manifestations Occurring in Syphilis and the Common Skin Diseases." Dr. Daniel P. Griffin will preside at the Section on Neurology and Psychiatry meeting and Dr. Harold G. Woolf, of New York City, will speak on "Migraine and Its Treatment." The meeting of the Connecticut Public Health Association will be presided over by Dr. Benjamin G. Horning and Dr. W. Bradford Walker, of Cornwall, will speak on "How Towns Can Organize for Public Health." At the meeting of the Hezekiah Beardsley Pediatric Club of Connecticut Dr. J. Harold Root will preside and Dr. Mitchell I. Rubin, of Philadelphia, will speak on "The Heart in Acute Glomerulo-Nephritis." Dr. Merrill K. Lindsay will preside over the Section on Orthopedic Surgery and Dr. A. Bruce Gill, of Philadelphia, will address the section on "Operative Treatment of Dislocations of the Hip."

On Thursday morning Dr. Joseph I. Linde, president-elect of the Connecticut State Medical Society, will preside. The following papers will be heard: "Rheumatic Fever," Dr. T. Duckett Jones, associate in medicine, Harvard Medical School; "Diagnosis and Treatment of Ulcerative Colitis," Dr. Z. Bercovitz, assistant clinical professor of medicine, Columbia University; "Thrombophlebitis in the Legs and Pelvis," Dr. John Homans, clinical professor of surgery, Harvard Medical School. Five-minute talks will be presented by Drs. Blake, Powers, Neuswanger, Nahum, Linde and Barker. In the afternoon there will be three symposia, "Rheumatic Fever" by Drs. Paul, Jones, Wilson, Salinger and Marvin; "Diagnosis and Treatment of Ulcerative Colitis" by Drs. Stringfield, Bercovitz, Buckstein, Carter, Clark and Fuller; "Thrombophlebitis in the Legs and Pelvis" by Drs. Oughterson, Homans, Bancroft, Lund and Winternitz.

The three-day courses are an innovation this year. These courses consist of co-ordinated lectures, demonstrations and discussions of special subjects throughout the three days of the Congress and will take place from 2:00 to 5:00 p. m. Courses are offered in the following subjects: "Contraception," Dr. Margaret Tyler; "Diagnostic Roentgenology," Drs. Hugh Wilson, Michael D'Amico and Lawrence Mucci; "Methods of Fracture Treatment Advocated for Fracture Surgeons," Dr. Merrill K. Lindsay, Dr. Robert M. Yergason, Dr. Charles W. Goffe, Dr. Arthur S. Griswold, Dr. J. L. Vickers; "Nervous System Changes in Anoxemia and Avitaminosis," Drs. H. M. Zimmerman, F. H. Lewy, James C. Fox, Jr.; "Recognition of Premalignant and Malignant Lesions of the Skin," Drs. Ellwood C. Weise, E. Myles Standish, Maurice J. Strauss and M. J. Morrissey.

Papers read before the congress will be published in subsequent issues of the *Journal of the Connecticut State Medical Society*. On Tuesday and Wednesday the congress will meet for a social hour at 364 Whitney Avenue, the home of the New Haven City Medical Association, from 5:00 to 6:30 p. m. The Women's Medical Society of Connecticut will meet for luncheon at the Dutch Treat Tea Room.

CARNEY HOSPITAL

The monthly clinical meeting and luncheon of the Carney Hospital will be held in the Andrew Carney Assembly Room, Monday, September 19, at 11:30 a. m.

Program: Case reports.

Physicians and students are cordially invited to attend.

ROY J. HEFFERNAN, M.D., *Secretary*.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held on Tuesday, September 27, in the Peter Bent Brigham Hospital amphitheater (Shattuck Street entrance), at 8:15 p. m.

PROGRAM

Presentation of cases.

The Diagnosis of Fevers in Practice. Dr. Philip Manson-Bahr, London, England.

Dr. Richard P. Strong will preside.

Medical students and physicians are cordially invited to attend.

ROBERT ZOLLINGER, M.D., *Secretary*.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, SEPTEMBER 19

MONDAY, SEPTEMBER 19

*11:30 a. m. Carney Hospital. Monthly clinical meeting and luncheon.

TUESDAY, SEPTEMBER 20

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

12 m. Massachusetts General Hospital. Surgical staff conference, amphitheater.

WEDNESDAY, SEPTEMBER 21

8 a. m. Massachusetts General Hospital. Grand rounds. Orthopedic department.

THURSDAY, SEPTEMBER 22

9 a. m. Massachusetts General Hospital. Surgical grand rounds.

9 a. m. Massachusetts General Hospital. Neurological staff clinic. Ether Dome.

*10:30 a. m. Psychiatric Rounds. Boston City Hospital. Rounds will start at Room 916 on the ninth floor of the Medical Building.

12 m. Massachusetts General Hospital. Clinicopathological conference.

FRIDAY, SEPTEMBER 23

10 a. m. Massachusetts General Hospital. Fracture rounds.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

SATURDAY, SEPTEMBER 24

*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.

SEPTEMBER 19 — Carney Hospital. Notice above.

SEPTEMBER 19-22 — Congress of the International Society of Surgery. Page 180, issue of August 4.

SEPTEMBER 19-23 — American Association of Railway Surgeons. Page 181, issue of August 4.

SEPTEMBER 20, 21 and 22 — Clinical Congress of the Connecticut State Medical Society. Page 408.

SEPTEMBER 26 and 27 — Symposium on Occupational Disease. Page 145, issue of July 28.

SEPTEMBER 27 — Harvard Medical Society. Notice above.

OCTOBER 3-15 — Inactive Status Training Course. Page 288, issue of August 25.

OCTOBER 4 and 5 — Massachusetts Medico-Legal Society. Page 365, issue of September 8.

OCTOBER 7 and 8 — Conference on Rural Medicine. Page 180, issue of August 4, and page 407 of this issue.

OCTOBER 8 and NOVEMBER 15 — American Board of Ophthalmology. Page 282, issue of February 10.

OCTOBER 13 — Pentucket Association of Physicians, 8:30 p. m., Hotel Bartlett, 95 Main Street, Haverhill.

OCTOBER 17, 1938 and FEBRUARY 20, 1939 — American Board of Internal Medicine, Inc. Page 216, issue of August 11.

OCTOBER 17-21 — Clinical Congress of the American College of Surgeons, New York City.

OCTOBER 24-26 — Academy of Physical Medicine, Scientific Session. Washington, D. C.

OCTOBER 24-NOVEMBER 4 — Graduate Fortnight of the New York Academy of Medicine. Page 181, issue of August 4.

OCTOBER 25-28 — American Public Health Association. Page 321, issue of September 1.

NOVEMBER 5 — American Board of Obstetrics and Gynecology. Page 145, issue of July 28.

MARCH 27-31, 1939 — American College of Physicians. Page 36, issue of July 7.

BOOK REVIEWS

Embryonic Development and Induction. Hans Spemann. 401 pp. New Haven: Yale University Press, 1938. $5.00.

Professor Hans Spemann, who was awarded the Nobel Prize in Medicine in 1935, records in this book a brilliant summary of work in which he himself has been actively engaged for more than a quarter of a century. In the introduction to these lectures, which were given at Yale University under the Silliman Foundation, the author points out that his purpose is "to trace the evolution of the problem in question in the clearest, simplest and most objective manner possible, to indicate the directions in which their solution has been attempted, and to set forth the final results which the search had hitherto attained." This purpose is admirably achieved in all these particulars.

Here is a book packed full of experiments with life studied as life, based on the initial achievement in this field by Wilhelm Roux. Considering the constant flow of new and often conflicting reports from various laboratories, the author merits our gratitude in placing before students of biology a book which not only will remain a landmark in the history of embryonic development and induction but will also serve as a beacon to guide future research students who choose to travel this direction.

Beginning with a discussion of the normal development of the amphibian egg, the author passes on to a consideration of the early experiments by Roux, Pflüger and Dreisch and of their effect upon the germ-plasm theory of Weismann. A chapter is then devoted to the development of the vertebrate eye, followed by chapters dealing with experiments on "potency," "determination," "organizers," "induction" and "embryonic field" and finally a discussion of the gradient theory.

This book is profusely illustrated and contains extensive reference to the literature. The ordinary and the controversial aspects are succinctly presented and make stimulating reading. This book will undoubtedly become an indispensable tool to all investigators.

The Life of Chevalier Jackson: An autobiography. 229 pp. New York: The Macmillan Company, 1938. $3.50.

This is one of the outstanding autobiographies of modern times and can hardly fail to interest doctors throughout this country and abroad. The author, born in Pittsburgh in 1865, depicts his life as a boy in a small school in the steel district of Pennsylvania. He was shy, rather unsociable and not interested in the usual things that boys like. From an early age he worked with his hands doing woodwork and painting. With no intimate friends and few companions, his life was one of torment and torture by the other children. He writes vividly of the cruelty which he underwent at that period of his life, a chapter reminiscent of the early life of Kipling.

Jackson had to earn his way through school and did this by decorating china and glass, even while studying medicine at Jefferson Medical College. He had distinct ability along these lines, later brought out by some of his exquisite paintings, reproductions of which are found in his book. Living alone in a small room and cooking his own meals, he passed through the years of his medical-school education in Philadelphia, selling books from house to house throughout New England during the summer. One year he went cod fishing from Gloucester, and when the cook on the boat misbehaved, young Jackson was chosen to take his place, much to the joy of all hands, for he knew how to prepare foods in a tasty manner. His ability as a cook he inherited from his French ancestors.

After graduating from Jefferson Medical College, he went to Europe in the steerage to see Morell Mackenzie, but he does not tell much about his adventures in London. Returning to Pittsburgh, he began practice in 1887 and did some of the early work on tonsillectomy. Partly because of his interest in laryngology he began working with the esophagoscope about 1890, and this instrument he perfected within the next ten years. After that he took up work on the bronchoscope and by 1900 had become an authority on diseases of the larynx, recognized not only in this country but abroad.

Jackson was called to Philadelphia in 1916 and there he has remained. At one time he held five different professorships in Philadelphia medical colleges, an unusual distinction which has probably never come to any individual before. His work on bronchoscopy is widely recognized but less perhaps is known of his fight for life, for he first developed pulmonary tuberculosis in 1911 and had three severe attacks before 1920.

One of his endeavors of great importance in preventive medicine was the drawing up and finally seeing through Congress of the Caustic Act to prevent the lye burns of the esophagus, particularly in children. This act, requiring the labeling of all containers of lye, was finally passed in 1927.

Jackson writes his autobiography with modesty and humility. His greatness, however, stands out on every page. Devoting his life to the cause of making bronchoscopy safe throughout the world, he has left an imprint upon American medicine that can never be erased.

L'Année Thérapeutique: Médications et Procédés Nouveaux. A. Ravina. 203 pp. Paris: Masson et Cie, 1938. 25 Fr. fr.

This French annual is on exactly the same order as our familiar American yearbooks; it consists of abstracts from various European and American journals together with the necessary editorial comment. Of the high quality of Dr. Ravina's work there can be no question. His book, however, lacks the range of its American counterpart; to cover about fifty subjects some seventy-five articles have been abstracted. The prospective buyer therefore should riffle it through as to the applicability of its topics; he need not fear as to the reliability of its contents.

The Harvey Lectures: Delivered under the auspices of the Harvey Society of New York, 1937-1938. Series XXXIII. 275 pp. Baltimore: The Williams & Wilkins Company, 1938. $4.00.

The Harvey Lectures, as usual, cover a wide field of medicine — from the nature of the visual process to experimental hypertension. Of particular interest are contributions by Lundsgaard, of Copenhagen, on muscle metabolism, Drinker, of Boston, on the lymphatic system, Stanley, of Princeton, on virus proteins and Koch, of Chicago, on sex hormones. In general this series of lectures cover the most advanced fields of medicine and reflect the highest type of research scholarship.

In the Name of Common Sense: Worry and its control. Matthew N. Chappell. 192 pp. New York: The Macmillan Company, 1938. $1.75.

This book reports the personal experiences of a psychologist, not a physician, who treats patients in mild psychoneurotic states, of tension, anxiety and worry, by psychotherapeutic methods. The author appears to be successful in this type of practice. He is well grounded in psychology but weak in medicine. The book cannot be recommended.